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President : G. A. Syme, M.B., M.S. (Melb.), Melbourne.

Vice-Presidents :
W. Anstey Giles, M.B., C.M. (Edin.), Adelaide.
T. C. Savage, M.B. (Lond.), F.R.C.S. (Eng.), Auckland, N.Z.
F. H. V. Voss, F.R.C.S., L.S.A. (Lond.), Rockhampton, Q.
George E. Clemons, M.D., C.M. (Edin.), Launceston.

Committee : Drs. G. H. Abbott, R. Steer Bowker, Thomas Fiaschi, H. C. V. Hinder, H. L. Maitland.

Secretary : Dr. Gordon Craig.

3. SECTION OF OBSTETRICS AND GYNÆCOLOGY.

President : G. Rothwell Adam, M.D. (Melb.), C.M. (Edin.), Melbourne.

Vice-Presidents :
J. A. Hamilton, B.A., M.B., L.R.C.S. (Irel.), Adelaide.
D. Hardie, M.D., C.M. (Aber.), Brisbane.
Felix Meyer, M.D. (Melb.), Melbourne.
B. M. Moorehouse, M.B., C.M. (Edin.), M.R.C.S., Christchurch, N.Z.

Committee : Drs. George Armstrong, Fourness Barrington, J. Foreman, E. T. Thring, Ralph Worrall.

Secretaries : Drs. W. H. Crago and Taylor Young.

4. SECTION OF ANATOMY, PHYSIOLOGY AND PHARMACOLOGY.

President : E. H. Embley, M.D., C.B. (Melb.), Melbourne.

Vice-Presidents :

W. A. Osborne, M.B. (R.U.I.), D.Sc. (Melb.), Melbourne.
Professor R. J. A. Berry, M.D., C.M. (Edin.), F.R.C.S., F.R.S.E., Melbourne.
Professor J. H. Scott, M.D., C.M., Otago, N.Z.

Committee : Dr. F. P. Sandes, Professor Anderson Stuart, Professor J. T. Wilson.

Secretaries : Drs. H. G. Chapman and S. A. Smith.

5. SECTION OF PATHOLOGY, BACTERIOLOGY, AND TROPICAL MEDICINE.

President : Wilton W. Love, M.B., C.M. (Edin.), Brisbane.

Vice-Presidents :

A. Breinl, M.D. (Prague), Townsville, Q.
W. S. Roberts, M.R.C.S. (Eng.), Dunedin, N.Z.
R. J. Bull, M.D., B.S. (Melb.), University, Melbourne.
W. R. Cavenagh-Mainwaring, M.B., B.S. (Adel.), F.R.C.S. (Eng.), Adelaide

Committee : Drs. J. B. Cleland, Sydney Jamieson, F. Tidswell, Prof. D. A. Welsh.

Secretary : Dr. E. V. Barling.

6. SECTION OF PUBLIC HEALTH AND STATE MEDICINE.

President : J. S. Purdy, M.D., C.M. (Aber.), Hobart.

Vice-Presidents :

B. Burnet Ham, M.D. (Brux.), M.R.C.S. (Eng.), L.R.C.P. (Lond.), D.P.H. (Cantb.), Melbourne.
T. Borthwick, M.D., C.M. (Edin.), Adelaide.
J. H. L. Cumpston, M.B., B.S. (Melb.), D.P.H., Melbourne.

Committee : Drs. R. Dick, R. J. Millard, T. W. Sinclair, Ashburton Thompson, and C. S. Willis.

Secretary : Dr. W. G. Armstrong.

7. SECTION OF OPHTHALMOLOGY.

President : J. W. Barrett, C.M.G., M.D., M.S. (Melb.), Melbourne.

Vice-Presidents :

C. F. G. Webster, M.B., B.S. (Melb.), Wellington, N.Z.
A. M. Morgan, M.B., C.B. (Adel.), Adelaide.
A. E. Martin, M.D. (Durh.), F.R.C.S. (Eng.), L.R.C.P. (Lond.), Perth, W.A.

Committee : Drs. R. H. Jones, Gordon MacLeod, Odillo Maher, Roland Pope.

Secretary : Dr. J. C. Halliday.

8. SECTION OF PSYCHOLOGICAL MEDICINE AND NEUROLOGY.

President : W. Beattie Smith, F.R.C.S. (Edin.), Melbourne.

Vice-Presidents :

W. Ernest Jones, M.R.C.S. (Eng.), Melbourne.
W. H. Macfarlane, M.B. (Melb.), New Norfolk, T.
S. H. R. Montgomery, M.B., B.S., R.U. (Irel.), Claremont, W.A.
J. F. S. Hay, M.B., B.S. (Aberd.), Wellington, N.Z.

Committee : Drs. A. W. Campbell, J. Froude Flashman, Chisholm Ross, Eric Sinclair.

Secretary : Dr. A. Davidson.

9. SECTION OF DISEASES OF CHILDREN.

President : A. Jeffreys Wood, M.D., B.S. (Melb.), Melbourne.

Vice-Presidents :

C. H. Officer, M.B. (Melb.), Perth, W.A.
P. B. Bennie, M.A., M.D., B.S. (Melb.), Melbourne.
R. D. Campbell, M.B., B.S. (Glas.), Hobart.

Committee : Drs. E. H. Binney, C. P. B. Clubbe, Macdonald Gill, E. S. Littlejohn.

Secretaries : Drs. W. F. Litchfield and R. B. Wade.

10. SECTION OF NAVAL AND MILITARY—MEDICINE AND SURGERY.

President : Col. James R. Purdy, M.D., C.M. (Aberd.), Director of Medical Service, New Zealand Defence Force, Wellington, N.Z.

Vice-Presidents :

Lt.-Col. A. Sutton, M.R.C.S. (Eng.), Brisbane.
Major A. F. White, V.D., L. & L.M.R.C.P., L.R.C.S. (Edin.), L.A.H. (Dubl.), Perth, W.A.

Committee : Lt.-Col. W. L'E. Eames, C.B. ; Lt.-Col. Perkins, D.S.O. ; Col. R. E. Roth, D.S.O.

Secretary : Major B. J. Newmarch.

11. SECTION OF DERMATOLOGY, RADIOLOGY, AND MEDICAL ELECTRICITY.

Presidents :

H. F. Lawrence, M.R.C.P. (Edin.), Melbourne.
F. J. Clendinnen, M.D. (Brux.), L.R.C.P. (Lond.), Melbourne.

Vice-Presidents :

E. W. J. Ireland, M.B., C.M. (Edin.), Hobart.
John Cuthbert, L.R.C.S. (Irel.), L.R.C.P. (Edin.), L.M.K. & Q.C.P. (Irel.), Perth, W.A.

Committee : Drs. C. Ayres, F. A. Bennet, L. Johnston.

Secretaries : Drs. W. McMurray and Herschel Harris.

12. SECTION OF LARYNCOLOGY, RHINOLOGY, AND OTOLOGY.

President : W. L. Robertson, M.B., C.M. (Edin.), Brisbane.

Vice-Presidents :

C. L. M. Iredale, M.R.C.S. (Eng.), Melbourne.
G. A. Fischer, M.B., B.S. (Melb.), Adelaide.
D. H. Lines, M.B., B.S. (Melb.), Hobart.

Committee : Drs. A. J. Brady, J. B. Graham, P. J. Kenna, T. S. Kirkland, H. J. Marks.

Secretary : Dr. Russell Nolan.

STATE SECRETARIES OF CONGRESS.

VICTORIA : Arthur Jeffreys Wood, M.D., B.S. (Melb.), 19 Collins-street, Melbourne.

QUEENSLAND : Wilton W. Love, M.B., C.M. (Edin.), Wickham Terrace, Brisbane.

SOUTH AUSTRALIA : John B. Gunson, M.B. (Adel.), M.R.C.S. (Eng.), North Terrace, Adelaide.

TASMANIA : Gregory Sprott, M.D., C.M., D.P.H. (Glas.), Macquarie-street, Hobart.

WEST AUSTRALIA : A. J. H. Saw, M.A., M.B., B.S., M.D., F.R.C.S. (Edin.), Perth, West Australia. *Acting Secretary :* W. Trethowan, M.B., M.S. (Aber.), Perth, West Australia.

NEW ZEALAND : South Island—Stanley Batchelor, F.R.C.S. (Eng.), L.R.C.P. (Lond.), Dunedin. North Island—W. E. Herbert, M.B., C.M., M.D. (Edin.), F.R.C.S. (Edin.), Boulcott-street, Wellington.

LIST OF PAST PRESIDENTS AND VICE-PRESIDENTS OF SECTIONS.

MEDICINE.

	Presidents.	*Vice-Presidents.*
1st Session, 1887 (Adelaide).	Dr. John Williams	Nil.
2nd Session, 1889 (Melbourne).	Dr. W. F. Taylor	Dr. D. Colquhoun, N.Z. Dr. J. D. Thomas, S.A.
3rd Session, 1892 (Sydney).	Dr. Jas. Robertson	Dr. J. C. Verco, S.A. Dr. E. O. Giblin, Tas. Dr. Colquhoun, N.Z. Dr. J. P. Ryan, Vic.
4th Session, 1896 (New Zealand).	Dr. A. A. Lendon, S.A.	Dr. Jas. Jamieson, Vic. Dr. Grant, Vic. Dr. J. L. Gibson, Q. Dr. Scot Skirving, N.S.W.
5th Session, 1899 (Brisbane).	Dr. Scot Skirving, N.S.W.	Dr. W. T. Hayward, S.A. Dr. J. W. Springthorpe, Vic. Dr. E. Jenkins, N.S.W. Dr. Colquhoun, N.Z. Dr. W. G. Maddox.
6th Session, 1902 (Hobart).	Dr. J. Jamieson, Vic.	Dr. Jarvie Hood, N.S.W. Dr. Walter Fell, N.Z. Dr. G. T. Howard, Vic. Dr. H. Swift, S.A. Dr. P. Bancroft, Q. Dr. T. H. Lovegrove, W.A.
7th Session, 1905 (Adelaide).	Dr. Colquhoun, N.Z.	Dr. G. E. Rennie, N.S.W. Dr. D. Grant, Vic. Dr. A. J. Turner, Q. Dr. H. Astles, W.A. Dr. R. R. Wislaw, Tas. Dr. W. Fell, N.Z.
8th Session, 1908 (Melbourne).	Dr. G. E. Rennie, N.S.W.	Dr. E. Roberton, N.Z. Dr. R. H. Martin, S.A. Dr. P. Bancroft, Q. Dr. Gregory Sprott, Tas.
9th Session, 1911 (Sydney).	Dr. W. T. Hayward, S.A.	Dr. H. C. Maudsley, Vic. Dr. J. G. Wolfhagen, Tas. Dr. T. L. Anderson, W.A. Dr. F. Goulburn Gibson, N.Z.

SURGERY.

	Presidents.	*Vice-Presidents.*
1st Session, 1887 (Adelaide).	Dr. T. N. Fitzgerald, Vic.	Nil.
2nd Session, 1889 (Melbourne).	Dr. E. C. Stirling, S.A.	Dr. C. H. Haines, N.Z. Dr. H. W. Maunsell, N.Z. Dr. Richard Rendle, Q.
3rd Session, 1892 (Sydney).	Dr. W. Gardener	Dr. J. W. Barrett, Vic. Dr. W. E. Collins, N.Z. Dr. F. A. Bowen, Vic. Dr. F. D. Bird, Vic. Dr. C. S. Ryan, Vic. Dr. Hardie, Q. Dr. A. Jamieson, W.A. Dr. J. Thomson, Q.

SURGERY—*continued.*

	Presidents.	*Vice-Presidents.*
4th Session, 1896 (New Zealand).	Dr. H. M. O'Hara, Vic.	Dr. A. MacCormick, N.S.W. Dr. W. A. Giles, S.A. Dr. J. Thomson, Q. Dr. S. Knaggs, N.S.W. Dr. Jas. Jackson, Vic.
5th Session, 1899 (Queensland).	Dr. A. MacCormick, N.S.W.	Dr. W. A. Giles, S.A. Dr. W. Moore, Vic. Dr. C. Clubbe, N.S.W. Dr. G. Cleghorn, N.Z. Dr. R. S. Bright, Tas.
6th Session, 1902 (Tasmania).	Dr. L. E. Barnett, N.Z.	Dr. W. Chisholm, N.S.W Dr. G. A. Syme, Vic. Dr. Hope Lewis, N.Z. Dr. F. Tratman, W.A. Dr. G. H. Hopkins, Q. Dr. B. Poulton, S.A.
7th Session, 1905 (Adelaide).	Dr. F. D. Bird, Vic.	Dr. H. C. Hinder, N.S.W. Dr. R. H. Russell, Vic. Dr. E. Jackson, Q. Dr. F. Tratman, W.A. Dr. R. G. Scott, Tas. Dr. W. E. Collins, N.Z.
8th Session, 1908 (Melbourne).	Dr. B. Poulton, S.A.	Dr. H. C. Hinder, N.S.W. Dr. E. S. Jackson, Q. Dr. W. Trethowan, W.A. Dr. John Ramsay, Tas.
9th Session, 1911 (Sydney).	Dr. G. A. Syme, Vic.	Dr. W. Anstey Giles, S.A. Dr. T. C. Savage, N.Z. Dr. F. H. V. Voss, Q. Dr. George E. Clemons, Tas.

OBSTETRICS AND GYNÆCOLOGY.

	Presidents.	*Vice-Presidents.*
1st Session, 1887 (Adelaide).	Dr. J. Foreman, N.S.W.	Nil.
2nd Session, 1889 (Melbourne).	Dr. F. C. Batchelor, N.Z.	Dr. R. S. Bright, Tas. Dr. Jas. Hill, Q. Dr. E. W. Way, S.A.
3rd Session, 1892 (Sydney).	Dr. W. Balls Headley, Vic.	Dr. F. C. Batchelor, N.Z. Dr. R. D. Pinnock, Vic. Dr. R. S. Bright, Tas. Dr. E. Hinchcliffe, Vic. Dr. E. W. Way, S.A.
4th Session, 1896 (New Zealand).	Dr. E. W. Way, S.A.	Dr. R. S. Bryant, Tas. Dr. F. H. Meyer, Vic. Dr. Ralph Worrall, N.S.W. Dr. C. F. Marks, Q.
5th Session, 1899 (Brisbane).	Dr. W. W. O'Sullivan, Vic.	Dr. A. A. Hamilton, S.A. Dr. J. A. G. Hamilton, S.A. Dr. G. R. Adam, Vic. Dr. E. T. Thring, N.S.W. Dr. T. M. Hocken, N.Z. Dr. J. E. Wolfhagen, Tas.

OBSTETRICS AND GYNÆCOLOGY—*continued.*

	Presidents.	*Vice-Presidents.*
6th Session, 1902 (Tasmania).	Dr. R. Worrall, N.S.W.	Dr. H. C. Taylor Young, N.S.W. Dr. F. W. W. Morton, Vic. Dr. A. F. Davenport, Vic. Dr. A. J. H. Saw, W.A. Dr. F. G. Connolly, Q.
7th Session, 1905 (Adelaide).	Dr. W. S. Byrne, Q.	Dr. E. T. Thring, N.S.W. Dr. G. R. Adam, Vic. Dr. T. G. Connolly, Q. Dr. H. Horrocks, W. A. Dr. C. J. Pike, Tas. Dr. B. Moorehouse, N.Z.
8th Session, 1908 (Melbourne).	Dr. E. Thring, N.S.W.	Dr. Jas. A. G. Hamilton, S.A. Dr. H. F. Harvey, W.A. Dr. F. G. Connolly, Q. Dr. B. M. Moorehouse, N.Z.
9th Session, 1911 (Sydney).	Dr. G. Rothwell Adam, Vic.	Dr. J. A. G. Hamilton, S.A. Dr. D. Hardie, Q. Dr. Felix Meyer, Vic. Dr. B. M. Moorehouse, N.Z.

ANATOMY AND PHYSIOLOGY.

	Presidents.	*Vice-Presidents.*
1st Session, 1889 (Adelaide).	Nil.	Nil.
2nd Session, 1889 (Melbourne).	Prof. Stuart, N.S.W. Baron Von Mueller, Pharmacology.	Prof. Watson, S.A.
3rd Session, 1892 (Sydney).	Prof. H. B. Allen, Vic.	Prof. Watson, S.A. Prof. Scott, N.Z. Prof. Spencer, Vic. Dr. J. Lockhart Gibson, Q. Dr. E. C. Stirling, S.A.
4th Session, 1896 (New Zealand).	Prof. Watson, S.A.	Dr. J. W. Barrett, Vic. Dr. E. Hirschfeld, Q. Dr. W. L. Cleland, S.A. Dr. H. Maudsley, Vic. Prof. J. T. Wilson, N.S.W.
5th Session, 1899 (Brisbane).	Dr. R. H. Marten.	Prof. C. J. Martin, N.S.W. Prof. J. T. Wilson, N.S.W. Dr. W. S. W. Roberts, N.Z. Dr. G. H. Hogg, Tas.
6th Session, 1902 (Tasmania).	Prof. J. H. Scott, N.Z.	Dr. Sydney Jamieson, N.S.W. Dr. Ed. Jennings, N.Z. Dr. J. A. Wheeler, Q. Dr. W. R. Cavenagh-Mainwaring, S.A. Dr. W. P. Seed, W.A.
7th Session, 1905 (Adelaide).	Prof. Welsh, N.S.W.	Prof. J. T. Wilson, N.S.W. Dr. C. H. Mollison, Vic. Dr. J. A. Wheeler, Q. Dr. W. Trethowan, W.A. Dr. A. H. Clarke, Tas. Dr. W. S. Roberts, N.Z.
8th Session, 1908 (Melbourne).	Prof. Wilson, N.S.W.	Prof. Stuart, N.S.W. Prof. Watson, S.A. Prof. Malcolm, N.Z.
9th Session, 1911 (Sydney).	Dr. E. H. Embley, Vic.	Dr. W. A. Osborne, Vic. Prof. R. J. A. Berry, Vic. Prof. J. H. Scott, N.Z.

PATHOLOGY AND BACTERIOLOGY.

	Presidents.	*Vice-Presidents.*
2nd Session, 1889 (Melbourne).	Dr. W. C. Wilkinson, N.S.W.	Nil.
3rd Session, 1892 (Sydney).	Prof. Allen, Vic.	Nil.
4th Session, 1896 (New Zealand).	Prof. Watson, S.A.	Nil.
6th Session, 1902 (Hobart).	Prof. J. H. Scott, N.Z.	Nil.
8th Session, 1908 (Melbourne).	Dr. F. Tidswell, N.S.W.	Dr. W. R. Cavenagh-Mainwaring, S.A. Dr. W. S. Roberts, N.Z. Dr. W. W. Love, Q.
9th Session, 1911 (Sydney).	Dr. Wilton W. Love, Q.	Dr. A. Breinl, Q. Dr. W. W. Roberts, N.Z. Dr. R. J. Bull, Vic. Dr. W. R. Cavenagh-Mainwaring, S.A.

PUBLIC HEALTH AND STATE MEDICINE.

	Presidents.	*Vice-Presidents.*
1st Session, 1887 (Adelaide).	Nil.	Nil.
2nd Session, 1889 (Melbourne).	Dr. H. N. MacLaurin, N.S.W.	Dr. Alex. Johnson, N.Z. Dr. Thos. C. Smart, Tas. Dr. Alfred R. Waylen, W.A.
3rd Session, 1892 (Sydney).	Dr. H. T. Whittell, S.A.	Dr. D. A. Gresswell, Vic. Dr. D. Macgregor, N.Z. Dr. F. Ogston, N.Z. Dr. J. V. McCreery, Vic. Dr. W. F. Taylor, Q. Dr. C. E. Barnard, Tas. Dr. W. H. McFarland, Tas. Dr. A. R. Waylen, W.A.
4th Session, 1896 (New Zealand).	Dr. J. W. Springthorpe, Vic.	Dr. W. H. Goode, N.S.W. Dr. D. Hardie, Q. Dr. J. E. Neild, Vic. Dr. C. Ross, N.S.W.
5th Session, 1899 (Brisbane).	Dr. J. A. Thompson, N.S.W.	Dr. T. Borthwick, S.A. Dr. J. V. McCreery, Vic. Dr. Eric Sinclair, N.S.W. Dr. F. Ogston, N.Z. Dr. T. Hope Lewis, N.Z. Dr. E. J. Crouch, Tas.
6th Session, 1902 (Hobart).	Dr. Thos. Cherry, Vic.	Dr. F. Tidswell, N.S.W. Dr. B. B. Ham, Q. Dr. A. C. F. Halford, Q. Dr. T. M. Kendall, N.S.W. Dr. J. V. McCreery, Vic.
7th Session, 1905 (Adelaide).	Dr. W. G. Armstrong, N.S.W.	Dr. F. Tidswell, N.S.W. Dr. W. B. Smith, Vic. Dr. B. B. Ham, Q. Dr. E. Black, W.A. Dr. G. F. Read, Tas. Dr. J. N. Mason, N.Z.
8th Session, 1908 (Melbourne).	Dr. J. N. Mason, N.Z.	Dr. T. Borthwick, S.A. Dr. B. B. Ham, Q. Dr. J. S. C. Elkington, Tas.
9th Session, 1911 (Sydney).	Dr. J. S. Purdy, Tas.	Dr. B. Burnet Ham, Vic. Dr. T. Borthwick, S.A. Dr. J. H. L. Cumpston, Vic.

STATE MEDICINE.

	Presidents.	Vice-Presidents.
1st Session, 1887 (Adelaide).	Dr. H. T. Whittell, S.A.	Nil.
7th Session, 1905 (Adelaide).	Dr. G. A. Syme, Vic.	Sir P. S. Jones, N.S.W. Dr. And. Shields, Vic. Hon. Dr. C. F. Marks, Q. Dr. T. G. Davy, W.A. Dr. F. Ogston, N.Z.

EYE, EAR AND THROAT.

	Presidents.	Vice-Presidents.
1st Session, 1887 (Adelaide).	Nil.	Nil.
2nd Session, 1889 (Melbourne).	Dr. M. J. Symons.	Dr. C. M. Anderson, N.Z. Dr. A. J. Brady, N.S.W. Dr. Thos. Evans, N.S.W.
3rd Session, 1892 (Sydney).	Nil.	Nil.
4th Session, 1896 (New Zealand).	Dr. H. M. O'Hara, Vic. Dr. Jas. Jackson, Chairman, Vic.	Dr. Jas. Jackson, Vic.
5th Session, 1899 (Brisbane).	Dr. H. L. Ferguson, N.Z.	Dr. T. K. Hamilton, S.A. Dr. A. L. Kenny, Vic. Dr. A. J. Brady, N.S.W. Dr. W. Odillo Maher, N.S.W. Dr. F. Wallace Mackenzie, N.Z. Dr. C. E. Barnard, Tas.
6th Session, 1902 (Hobart).	Dr. T. K. Hamilton, S.A.	Dr. T. S. Kirkland, N.S.W. Dr. C. M. Iredale, Vic. Dr. W. M. Stenhouse, N.Z. Dr. E. L. Gault, Vic. Dr. A. Francis, Vic. Dr. Hy. T. Kelsall, W.A.
7th Session, 1905 (Adelaide).	Dr. A. J. Brady, N.S.W. Dr. Odillo Maher, N.S.W.	Dr. G. T. Hankins, N.S.W. Dr. P. S. Webster, Vic. Dr. J. L. Gibson, Q. Dr. H. T. Kelsall, W.A. Dr. W. W. Giblin, Tas. Dr. H. L. Ferguson, N.Z.
8th Session, 1908 (Melbourne).	Dr. J. Lockhart Gibson, Q.	Dr. F. A. Pockley, N.S.W. Dr. G. H. Hogg, Tas. Dr. G. M. Scott, W.A.

OPHTHALMOLOGY.

	President.	Vice-Presidents.
9th Session, 1911 (Sydney).	Dr. J. W. Barrett, Vic.	Dr. C. F. G. Webster, N.Z. Dr. A. M. Morgan, S.A. Dr. A. E. Martin, W.A.

NEUROLOGY.

	Presidents.	Vice-Presidents.
2nd Session, 1889 (Melbourne).	Dr. F. N. Manning, N.S.W.	Dr. W. E. Hacon, N.Z. Dr. A. S. Paterson, S.A. Dr. R. R. Scholes, Q.
8th Session, 1908 (Melbourne).	Dr. Eric Sinclair, N.S.W.	Dr. W. L. Cleland, S.A. Dr. J. B. Hogg, Q. Dr. Chisholm Ross, N.S.W.
9th Session, 1911 (Sydney).	Dr. W. Beattie Smith, Vic.	Dr. W. Ernest Jones, Vic. Dr. W. H. Macfarlane, Tas. Dr. S. H. R. Montgomery, W.A. Dr. J. F. S. Hay, N.Z.

DISEASES OF CHILDREN.

	Presidents.	*Vice-Presidents.*
2nd Session, 1889 (Melbourne).	Dr. W. Snowball.	Nil.
8th Session, 1908 (Melbourne).	Dr. A. J. Turner, Q.	Dr. C. B. Clubbe, N.S.W. Dr. A. A. Lendon, S.A. Dr. A. E. Mills, N.S.W.
9th Session, 1911 (Sydney).	Dr. A. Jeffreys Wood, Vic.	Dr. C. H. Officer, W.A. Dr. P. B. Bennie, Vic. Dr. R. D. Campbell, Tas.

NAVAL AND MILITARY—MEDICINE AND SURGERY.

	Presidents.	*Vice-Presidents.*
8th Session, 1908 (Melbourne).	Surg. Gen. Williams.	Col. J. Thomson, Q. Lt.-Col. Fiaschi, N.S.W. Lt.-Col. Lantour, N.Z. Major W. Giblin, Tas.
9th Session, 1911 (Sydney).	Col. James R. Purdy, N.Z.	Lt.-Col. A. Sutton, Q. Major A. F. White, W.A.

SKIN, RADIO-THERAPATHY, &c.

	Presidents.	*Vice-Presidents.*
2nd Session, 1889 (Melbourne).	Dr. J. P. Ryan.	Nil.
8th Session, 1902 (Melbourne).	Dr. McMurray, N.S.W.	Dr. H. Herschel Harris, N.S.W. Dr. W. P. Seed, W.A. Dr. E. J. Roberts, Tas.
9th Session, 1911 (Sydney).	Dr. H. F. Lawrence, Vic. Dr. F. J. Clendinnen, Vic.	Dr. E. W. J. Ireland, Tas. Dr. John Cuthbert, W.A.

LARYNCOLOGY, RHINOLOGY, AND OTOLOGY.

	President.	*Vice-Presidents.*
9th Session, 1911 (Sydney).	Dr. W. N. Robertson, Q.	Dr. C. L. M. Iredale, Vic. Dr. G. A. Fischer, S.A. Dr. D. H. Lines, Tas.

LIST OF MEMBERS OF CONGRESS.

Abbott, G. H., M.B., M.S. (Syd.), Macquarie-street, Sydney.
Adam, G. Rothwell, M.D. (Melb.), M.B., C.M. (Edin.), F.R.S.M. & O.S. (Edin.), 84 Collins-street, Melbourne, Vic.
Adamson, John, M.B., C.B. (Melb.) Malvern, Vic.
Adey, J. K., M.B., B.S. (Melb.), Ballarat, Vic.
Agnew, J. F., M.B., C.M. (Glas.), Emu-street, Richmond, Vic.
Ahearne, J., L.R.C.S. (Irel.), Townsville, Q.
Ahearne, Joseph, L.R.C.S. (Ire.), L.R.C.P. (Lond.), Townsville, Q.
Aitchison, A. S., M.B., C.B. (Melb.), Albert Park, Melbourne, Vic.
Alcorn, R., L.R.C.P. (Edin.), L.R.C.S. (Irel.), West Maitland.
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Honorary Members.

Professor Haswell, Sydney University.
Professor Douglas Stewart, Sydney University.
Professor Fawcett, Sydney University.
Professor Pollock, Sydney University.
Professor J. A. Schofield, Sydney University.
H. D. Walsh, President, Royal Society, Sydney.
J. J. Fletcher, Linnean Society, Sydney.
Donald Smith, Dental Association, Sydney.
P. Board, M.A., Under Secretary, Public Instruction, N.S.W.
F. M. Bladen, Government Librarian, Sydney.
J. H. Maiden, Director, Botanic Gardens, Sydney.
J. B. Trivett, New South Wales Statistician.
W. M. Hamlet, Government Analyst, N.S.W.
W. A. Gullick, Government Printer, N.S.W.
W. Froggatt, Government Entomologist, N.S.W.
G. H. Knibbs, Commonwealth Statist, Victoria.
Harvey Johnson, Bureau of Microbiology, N.S.W.
G. P. Darnell Smith, Bureau of Microbiology, N.S.W.
H. P. Owen, Master-in-Lunacy, N.S.W.
R. Greig Smith, Woollahra, N.S.W.
Sydney Dodds, Sydney University.
O. C. Beale, Burwood, N.S.W.
A. W. Green, Assistant Secretary of Congress, Burwood, N.S.W.
F. B. Guthrie, Department of Agriculture, N.S.W.
P. Barrachi, Government Astronomer, Victoria.
President, Pharmaceutical Society, Sydney.
Government Astronomer, Sydney.

PROGRAMME OF MEETINGS AND ENTERTAINMENTS.

Saturday, September 16—

10 a.m. to 1 p.m. ...	Registration of Members at the B.M.A. Library, 30–34 Elizabeth-street, Sydney.—Distribution of Programmes and Invitations.
11 a.m.	Reception by Lord Mayor of representative Officers of Congress.
3.30 p.m.	Reception at University Grounds.—Registration of Members and distribution of Invitations at the Medical School, Sydney University.

Sunday, September 17—

At St. Andrew's Cathedral a special service was held at 11 a.m. The lessons were read by Archdeacons Boyce and Gunther. The Archbishop of Sydney (Dr. Wright) preached the sermon from the text taken from Galatians, chapter VI, verse 2—" Bear ye one another's burdens and so fulfil the law of Christ."

At St. Mary's Cathedral Father Sykes welcomed the members of Congress in the name of the Archbishop of Sydney (Dr. Kelly).

At St. Paul's, Redfern, Archdeacon Boyce at the 7 o'clock service, preached from the text—" Luke, the beloved Physician."

At the Stanmore Methodist Church the Rev. C. J. Prescott, Headmaster of the Newington College, preached a sermon dealing with the value of the medical profession, his text was " Luke, the beloved Physician."

At the Woollahra Presbyterian Church the Rev. Dr. Macqueen preached a sermon from the text " The right scientific spirit," from 1st Kings, chapter X, verse 1.

Monday, September 18—

9 a.m.	Registration of Members at Great Hall, Sydney University. Distribution of Programmes and Invitations.
9.30 a.m.	Business Meeting at Great Hall, Sydney University.
2 p.m.	Harbour Excursion (by New South Wales Government), s.s. " Kulgoa," from Fort Macquarie—East of Circular Quay—round Sydney Harbour, returning at 5.30 p.m.
7.30 p.m.	Organ Recital, Town Hall, Sydney, by Ernest Truman, Esq., City Organist.
8.30 p.m.	Inaugural Meeting—Opening of Congress by His Excellency the Governor-General—President's Address—at Town Hall, Sydney.

Tuesday, September 19—

8.30 a.m.	Demonstrations at Royal Prince Alfred, Sydney, St. Vincent's, Royal Alexandra, and the Lewisham Hospitals.
9 a.m.	Sectional Meetings at the Medical and Engineering Schools.
1 p.m.	Lunch in the Great Hall, Sydney University.
3.30 p.m.	Garden Party at State Government House, Rose Bay.
9 p.m.	Evening Party at the Sydney Town Hall (given by the President and Council of the N.S.W. Branch of the B.M.A.).

Wednesday, September 20—

8.30 a.m.	Demonstrations at Royal Prince Alfred, Sydney, St. Vincent's, Royal Alexandra, and the Lewisham Hospitals.
9 a.m.	Presidential Address in Section of Medicine by Dr. W. T. Hayward.
10 a.m.	Combined Discussion on Serum Therapy and Vaccine, The Great Hall, University—Professor Allen opened the discussion.
3.30 p.m.	Garden Party at Yaralla (tendered by Miss Walker). Steamers left the wharf at Macquarie Point at 2 o'clock.
7.30 p.m.	Four Sectional Dinners (given by the N.S.W. Members of Congress) at Paris House, Australian Club, Aaron's "Exchange Hotel," and the "Wentworth Hotel."

Thursday, September 21—

8.30 a.m.	Demonstrations at Royal Prince Alfred, Sydney, St. Vincent's, Royal Alexandra, and the Lewisham Hospitals.
9 a.m.	Sectional Meetings at the Medical and Engineering Schools.
1 p.m.	Lunch in the Great Hall, Sydney University.
Afternoon	Garden Parties and Motor Excursions.
3 p.m.	Garden Party at "Ginahgulla," Double Bay (given by Sir James and Lady Fairfax to some Members of Congress).
3 p.m.	Afternoon tea at the Royal Sydney Golf Club by some ladies of the Committee and other Members of the Golf Club.
9 p.m.	Ball given by President and Mrs. Antill Pockley, Town Hall, Sydney. The Governor-General and Lady Denman, also the Acting Governor and Lady Cullen, were present.

Friday, September 22—

8.30 a.m.	Demonstrations at Royal Prince Alfred, Sydney, St. Vincent's, Royal Alexandra, and the Lewisham Hospitals.
9 a.m.	Sectional Meetings at the Medical and Engineering Schools.
1 p.m.	Lunch in the Great Hall, Sydney University.
3.30 p.m.	Garden Party at Victoria Barracks (given by the Army Medical Corps).
Evening	Dinner at State Government House to Officers of Congress.
Evening	Ladies' Committee entertained Members at the Royal Yacht Club, North Sydney.

Saturday, September 23—

Morning	Final Business Meeting at 9 o'clock, at the B.M.A. Library, Sydney.
10 a.m.	Visit to Sanatoria, Blue Mountains. The President of the Queen Victoria Homes for Consumptives (Sir Philip Sydney Jones), and Dr. McIntyre Sinclair, the proprietor of "Bodington" Private Sanatorium at Wentworth Falls, invited twenty (20) members of Congress, interested in the Sanatorium treatment of consumption, with their wives and daughters to visit their Sanatoria on the Blue Mountains on Saturday, the 23rd September.

Saturday, September 23—*continued.*

2 p.m. ... ... Fifty members of Congress were taken from the Chief Secretary's Department, Sydney, in motor cars to inspect the Home for Consumptives at Waterfall. Afternoon tea was provided, and a pleasant afternoon was spent.

2 p.m. ... ... Launch "Psyche" left Fort Macquarie to take members of Congress (to the number of forty-five) at invitation of Inspector-General of Insane to Garden Party at Hospital for Insane, Gladesville.

2 p.m. ... ... Trip to Cronulla. Special Tram arranged to meet 2 p.m. train from Sydney, returning by 5.30 p.m. train from Sutherland. Afternoon tea was provided at "The Cabin," Cronulla, by Mrs. Gordon-Craig and Mrs. Charles Clubbe.

2.25 p.m. ... ... Excursion to Jenolan Caves given by the Government of N.S.W. to sixty visitors. (This number was inclusive of ladies.) Left Sydney by the 2.25 p.m. train from No. 2 Platform for Medlow Bath, arriving at 9.25 p.m. Stayed night at Hydro Majestic, Medlow.

Sunday, September 24—

9.30 a.m. ... ... Motor to Jenolan Caves, 3 p.m. and 7.30 p.m., inspection of Caves. Stayed night at Caves House.

Monday, September 25—

10 a.m. ... ... Inspection of Caves. 1.15 p.m. motor to Mount Victoria and joined the 4.7 p.m. train for Sydney, arriving at 6.52 p.m.

The following golf clubs made Members of Congress honorary members during their stay in Sydney:—Australian Golf Club, Kensington; Royal Sydney Golf Club, Rose Bay; Manly Golf Club; Killara Golf Club.

The Automobile Club of Australia enrolled motorist Members of Congress as honorary members during their stay in Sydney.

Any Member of Congress who would be eligible for membership of the University Club if resident in Sydney, was made an honorary member of the club during his stay in Sydney.

The Women's Patriotic Club and Queen Victoria Club made lady visitors honorary members.

The Australian Club and the Australian Jockey Club made visiting members honorary members of the clubs during their visit.

COMBINED SECTIONAL MEETINGS.

Tuesday, 19th September, 1911.

9 a.m.—Conjoint discussion with Sections of Pathology and Public Health on "The Prevention of Syphilis."

Discussion opened by B. Burnet Ham, M.D.

Drs. Konrad Hiller, G. Lamble, J. S. Purdy, J. Ashburton Thompson, Valintine, Millard, and others took part in the discussion.

Thursday, 21st September, 1911.

Discussion on Infantile Paralysis, held by the conjoint Sections of Diseases of Children, Neurology, Pathology, and Public Health, on the morning of Thursday, September 21st, in the Theatre.

Openers of the discussion—

"Pathology and Etiology," Wilton W. Love, M.B., C.M.
"Relationship to Public Health," J. S. Purdy, M.B., M.S.
"Diagnosis and Symptomatology," A. Jeffreys Wood, M.D., B.S.
"General Surgical Treatment," W. Kent Hughes, M.B.

The following members took part in the discussion:—Drs. Mackenzie, C. S. Willis, W. J. Stewart McKay, J. MacDonald Gill, Max Herz, E. H. Binney, W. F. Litchfield, and R. B. Wade.

DEMONSTRATIONS.

The Pathological Department of the Sydney Hospital was open for inspection throughout the day. Drs. Jamieson and Finckh were in attendance on Tuesday, Wednesday, Thursday, and Friday from 8.30 a.m. till 9.30 a.m.

Demonstrations were given on the following subjects:—

1. Wassermann Reaction.
2. Sahli's method of Tuberculin administration.
3. Ultra Microscope.
4. Vaccine manufacture.
5. Rapid sectioning method for use in operation theatre.

Demonstrations at Sydney Hospital. Thursday, 21st September, 1911.

Exhibits of rare skin diseases.

Friday, 22nd September, 9 *a.m.*

Dr. Langloh Johnston exhibited the Radium Plant and cases under treatment, also intra-venous administration of Salvarsan.

Demonstrations and operations took place at the Royal Prince Alfred, Sydney, St. Vincent's Hospitals between 8 and 9 a.m. Monday to Friday inclusive.

Demonstrations of cases of Infantile Paralysis and Surgical Tubercule: Tuesday, 20th September, at 9 a.m.

Demonstration at the Out-patient Department, Royal Alexandra Hospital, Valentine-street, Sydney, near Sydney Railway-station.

Clinical demonstration of Kindergarten methods as carried on at the Hospital for the Insane, Newcastle, was given by Dr. Eric Sinclair, at the Medical School, Sydney University, on Friday, 22nd September, at 11 a.m.

CONGRESS MUSEUM.

The Congress Museum, devoted solely to Pathological specimens, in the old Hunterian Theatre.

ANATOMICAL EXHIBITS.

Prof. J. T. Wilson demonstrated these in his large room, Medical School.

THE HOSPITALS FOR THE INSANE.

The Pathological Laboratory of the Lunacy Department at the University were open daily during Congress between 12 and 1 p.m. Drs. Flashman, Latham, and Campbell exhibited specimens and gave demonstrations.

The Hospitals for the Insane, Callan Park, Gladesville, Parramatta, Rydalmere, the Mental Hospital and the Reception House, Darlinghurst, were available for inspection during Congress.

ANTHROPOMETRIC COMMITTEE.

The Anthropometric Committee of the Australasian Association for the Advancement of Science met early in Congress week.

An Anthropometric exhibit was arranged of specimens, instruments, charts, photographs, &c., to illustrate modern Anthropometric research. Demonstrations of the method of measuring the living subject were given.

THE CONGRESS MUSEUM AND EXHIBITION.

At the Session of Congress held in Melbourne in 1908, there was an excellent Exhibition owing to the enterprise of various manufacturing firms and also an attractive Museum, which proved of great interest to the members present on that occasion.

Shortly after this session, applications were received from firms desirous of exhibiting surgical instruments and appliances, pharmaceutical products and foods, and bacteriological apparatus, and a Museum Committee was appointed to make the necessary arrangements. After some deliberation, it was decided that such exhibits could only be adequately accommodated in the spacious Drawing Office of the Engineering School of the University. Every application was placed before the Museum Committee and when approved, the intending exhibitor was so notified and invited to state definitely the amount of floor space required. Owing to the large number of applications and the amount of space requested, it was found necessary to curtail the latter in some instances, in order to enable all exhibitors to have the opportunity of showing products of interest to the medical profession. Indeed, the demand became so great that the approaches to the Drawing Office of the Engineering School were finally utilised. Later, the terms on which exhibits were received were sent to each approved exhibitor, bearing particulars as to the dates of placing exhibits in position, with other necessary details, and a plan drawn to scale showing the position of firms and amount of allotted space was provided and rendered accessible. The Committee rigidly excluded patent and proprietary articles, and reserved the right to veto—a right which on several occasions was enforced. The Museum Committee express their gratitude and thanks to the professorial and teaching staff of the Engineering School of the University of Sydney for their kindly co-operation in helping them to make use of their beautifully-equipped Drawing Office, which was almost new, well lighted, with spacious approaches, and for their generosity in allowing the constant current to be available for working electrical exhibits. Although many of the exhibits were the products of large manufacturers abroad, yet a considerable amount of the hospital furniture and many surgeons' and physicians' requisites were of local manufacture, and, judged by the appearance, the local-make can compare favourably with the imported article. The exhibiting firms were:—Burroughs, Wellcome, & Co., Parke, Davis, & Co., Faulding & Co., Fairchild Bros., Merck, Allen and Hanbury, Meyer, Meltzer and Jackson, Donald Ross & Co., Elliott Bros., Potter and Birks, Seabury and Johnson, Oppenheimer, Son, & Co., The Manhu Food Co., Hannams Ltd., Watson and Sons, Thom (bacteriological apparatus), Bishop (optician), Stirling & Co. (medical books), Loxton (medical directory). The best thanks of the Committee are due to these exhibitors who worthily did their share in making the exhibition a complete success.

The Committee decided to organise a Congress Museum devoted exclusively to the exhibit of pathological specimens, and further resolved, as one of its objects, that if any paper required demonstration by pathological specimens, these might be temporarily removed from the Museum. The old Hunterian Theatre in the Medical School, as being most appropriate, was chosen for this purpose. A large amount of pathological material was collected by medical men in the metropolitan area who were interested in the success of this exhibit, which, after careful preparation and mounting, was arranged in sections and each specimen appropriately described. In the endeavour to make the Congress Museum a real success, the Secretaries of all the Australian States and the Dominion of New Zealand were by letter requested to invite visiting members to bring pathological material for exhibition. The Museum Committee is grateful to those members of Congress who responded, and to all who kindly co-operated by contributing the results of their own work, and to whom in a large measure the success of the Museum was due. Dr. Herman Lawrence showed a series of original, highly artistic and beautiful moulages, illustrating diseases of the skin, and Dr. Clenning Fenwick, similar models recording the effects of treatment of X-rays burns. Dr. Herschel Harris exhibited his collection of renal calculi skiagrams with the removed calculi attached, and a series of plaster casts of radium cases, before and after treatment. The Government Bureau of Microbiology, Sydney, under the directorship of Dr. Tidswell, sent numerous pathological specimens from the lower animals, a collection of cestode and nematode worms from man and other hosts, a series of botanical specimens showing the effects of parasites, and an exhibit illustrating the biochemical method of separation of colon-typhoid, paratyphoid, and dysentery type of organisms. Dr. Scholes showed a diphtheritic cast (which under antitoxin treatment was coughed up in one continuous piece) of the larynx, trachea, brochi and both lungs down to the finest bronchioles. Dr. Rothwell Adam's recent specimen of primary ovarian pregnancy mounted in formalin-gelatin was a perfect example of this very rare condition. Dr. Breinl showed a number of very interesting specimens from the Australasian Institute of Tropical Medicine.

The interest of the Congress Museum was greatly enhanced by the close proximity of the excellent Museums of the Anatomical and Pathological Departments which were thrown open by the Professors to the Congress. The Professors of the Medical Faculty personally conducted members of Congress through the departments under their control, giving much appreciated demonstrations of their valuable, comprehensive and instructive contents.

The one small regret that can alone be expressed was that the pressure of sectional meetings and social functions prevented many members from visiting the Exhibition and the Museum and left insufficient time for the greater number, who saw and appreciated the contents, to devote to them the careful inspection they merited.

EXHIBITS.

The Drawing Office of the Engineering School at the Sydney University was entirely set apart for the general exhibits, which attracted considerable attention throughout the week.

Messrs. Burroughs, Wellcome, & Co. had an attractive exhibit. Compressed medicaments were tastefully displayed in specially designed cases. Many of the drugs exhibited have been added to the physician's armamentarium by the investigations of scientific experts in the firm's laboratories at Dartford, England. Epinine being the most recent addition. Lodal and Ernutin were also displayed. They showed a new gauze—" Tabloid " Bismuth Gauze—odourless and non-toxic. Among other objects which attracted attention was a display of medicine-chests and cases. A series of photographs showing various scenes on the firm's materia medica farm, interesting charts, and kymographic tracings were also on view.

Parke, Davis, & Co.'s display consisted entirely of scientific products—Serums, Vaccines, Tuberculins, Biological products, Andrenalin preparations, Physiologically Standardised products, such as Ergot, Digitalis, Strophanthus, Cannabis, Indica, &c.; Sterilised Ampoules for hypodermic use, and Standardised Fluid Extracts. Among their new products the following were prominent:—Oculets, Ampoules Sodium Cacodylate, Ampoules Quinine and Urea Hydrochloride, the new local anæsthetic, Globules Stearosan, Bismuth Paste, and Capsules of Dissicated Corpora Lutea. The wall surrounding the exhibit was hung with interesting pictures, giving views of the biological and bacteriological departments at Detroit, U.S.A., showing the scientific methods of manufacturing serums, vaccines, tuberculins, &c.

E. Merck's special pharmaceutical preparations were prominently and tastefully displayed, and attracted considerable attention. The principal lines shown were Bromipin, Dionin, Fibrolysin, Iodipin, Jequiritol and Jequiritol Serum, Magnesium-Perhydrol, Perhydrol, Proponal, Stypticin, Tannoform, Veronal, Veronal Sodium.

Manhu Diabetic Foods (starch changed), manufactured by the Manhu Food Co., Ltd., Liverpool, were tastefully displayed by the Australian representative, Mr. Charles E. Hall, of Melbourne, and were favourably commented on by the visiting doctors.

F. H. Faulding & Co. had an attractive display of their Solyptol Antiseptic Soap, Solyptol Antiseptic Liquid, Creodol, Lactone Buttermilk Tabulets, Aldeform Tabulets, and Milk Emulsion. For all of these preparations Fauldings were successful in obtaining gold medals at the Franco-British Exhibition. They prominently exhibited their medals, effectively mounted on blue plush, in an oaken frame.

Messrs. Muir and Neil exhibited the products of Oppenheimer, Son, & Co., Ltd., for whom they are agents, special prominence being given to their Bi-Platinoids and Seawater Plasma, collected 200 miles from land off the coast of England, under the aseptic conditions required by Dr. Robt. Simon, of Paris; Pulverettes, Roboleine, and an Acid Pepsin and Bismuth Solution under the name of Liquor " Gastrorube," were also shown.

Jeyes' Sanitary Compounds were set out to good effect.

Messrs. Fassett and Johnson, agents for Seabury and Johnson, had a good display of medicinal and surgical plasters, absorbent and antiseptic cottons, surgical dressings, &c. Belladonna, Mead's Rubber Adhesive Plaster, " Standard " Gauzes, Mead's Moist Gauzes, B.W.B. Elastic Woven Bandage, Seabury's Absorbent Cottons and Ligatures, Portable Accident Cases, Sulphur Candles, " Perfection " Formaldehyde Fumigator, and Sanitary Cuspidore (or Spitting Cup) were the principal lines.

Messrs. Potter and Birks, Ltd., managing agents in Australasia for Johnson and Johnson, Surgical Dressing Manufacturers, and the J. Ellwood Lee Co., manufacturers of Trusses and Surgical Rubber goods, exhibited specimens of the products of these two houses. Samples of surgical catgut, plain and chromic, silkworm gut, catheters, hypodermic syringes, cotton goods for surgical use, maternity outfits, bandages, gauze strips for packing, synol liquid antiseptic soap, camphenol, liquid disinfectant and antiseptic, Bellevue rolls, &c., were shown.

Allen and Hanbury, Ltd., had an interesting and up-to-date display of surgical instruments, electrical appliances, &c., all of which are manufactured at their own factory at Bethnal Green, London. Their raw catgut and operating gloves and the familiar Allenbury's foods were also in prominence.

Elliott Bros., Ltd., exhibited surgical instruments and appliances and hospital furniture, a good portion of the latter being of local manufacture. Amongst the instruments which attracted attention was a carbon dioxide apparatus for the production of carbonic snow for application in the treatment of lupus, nævi, and tracoma. They also showed Martin's modification of the Riva Rocca Sphygomanometer, and a new needle for the intravenous injection of Salvarsan.

Hannams, Ltd., had on view their latest manufactures in operating appliances, sterilising plant, &c., for which they received a gold medal at the Franco-British Exhibition. The operating table they exhibited commanded a good deal of attention, its special features being an adjustable metal block for liver and kidney work, a simple warming apparatus, and an arrangement by means of which the whole table can be raised off the castors to suit various heights of operators. The sterilising plant consisted of apparatus for the sterilisation of water, dressings, instruments, catgut, &c., ward trolleys, metal screens, ward lockers, irrigator, lotion stands, instrument cabinets, towel stands, &c., combined to make an effective and interesting display.

Donald Ross & Co. had a splendid display of surgical instruments and appliances, from an enamel pus basin or stethoscope to an X-ray machine or a convertible baloptican for lantern, with opaque and microscopical projection. They also had on view a good display of hospital furniture, comprising operating tables, cabinets, wheel stretchers, solution bowls, dressing tables, dressing trolleys, and the Rochester steriliser. The vaccines from the Wimpole Vaccine Institute, Harley-street, London, attracted a good deal of attention. They also had on view the Bausch and Lomb microscopes.

The most prominent feature in W. Watson & Sons' exhibit was their "Sunic" X-ray outfit, designed especially for taking instantaneous radiographs. A current from the main was connected up with the exhibit to enable demonstrations of the working of the apparatus to be given. The novel features of the equipment consisted of the oak cabinet containing the whole of the apparatus—intensified coil, switchboard motor interrupter, time switch, milliampere meter, valve tube, &c. A new intensifier screen and a new tube stand, provided with universal movement and counterpoise to admit of easy raising and lowering of the X-ray tubes, with its lead glass shield, was also shown. Several tubes of special design for quick radiography and for therapeutic work were shown, together with various appliances for the protection of the operator. This firm also exhibited radium bromide and various applicators for therapeutic purposes—the Kiel radium tablets for making radio-active baths, and water for inhalation and drinking. Their display of microscopes with various accessories, including the new condenser for viewing living bacteria, telescopes and science lanterns, combined to present an attractive and interesting display.

Messrs. Meyer and Meltzer, of Melbourne, also had a splendid collection of surgical instruments and appliances, and Mr. F. E. Bishop exhibited the latest ophthalmic instruments.

An excellent exhibit, consisting entirely of microbiological apparatus, was that of Mr. Andrew Thom, of George-street, Sydney, every item in the interesting and varied display being manufactured in Australia—a fact which many of the visiting doctors could hardly believe, the scientific apparatus being quite up to the standard of the best manufactured abroad. Doctors interested in culture tests and students of bacteriology were greatly impressed by this display.

Messrs. Stirling & Co., of Melbourne, had a bookstall replete with the latest medical, surgical, and dental publications.

CATALOGUE OF EXHIBITS OF THE GOVERNMENT BUREAU OF MICROBIOLOGY, SYDNEY.

1. *Necrosing dermatitis. Head of sheep.* (358.)

Sheep's head with symmetrical necrosing dermatitis. This is perhaps the condition known as "fagopyrismus," due to the ingestion of certain kinds of food and subsequent exposure to sunlight. In this condition only non-pigmented areas are affected.

2. *Tuberculosis of Retro-pharyngeal glands. Cow.* (37.) The small gland normal.

Enormously enlarged tubercular gland with one of normal size for comparison. The gland is full of pus. Its size is considerably bigger than that of an infant's head.

3. *Tuberculosis. Retro-pharyngeal glands. Cow.* (76.)

A very large fibrous gland, showing throughout caseating white tubercular areas.

4. *Tuberculosis of neck glands. Dairy cow.* (248.)

Old lesions, fibrotic and calcifying.

5. *Tuberculosis. Glands of neck. Human.* (622.)

Many tubercle bacilli.

6. *Tuberculosis of Eye. Calf.* (637.)

Sections show in places vast masses of tubercle bacilli.

7. *Tuberculosis of Kidney. Bullock.* (7).

The small whitish projecting nodules are tubercles. Note that some of the kidney lobules are much more affected than others.

8. *Tuberculosis invading Muscle (ham). Pig.* (36.)

Notice the large, yellowish-white, nodular masses of tubercles.

9. *Tuberculosis of Hoof. Bullock.* (1.)

Hoof of bullock disorganised by tuberculosis, which appears as yellowish-white areas throughout the section.

10. *Actinomycosis of Udder. Cow.* (652.)

The lesions appear as yellowish-white nodules, often projecting into the milk ducts and sinuses, accompanied by fibrosis.

11. *Actinomycosis of Hoof. Cow.* (639.)

Much disorganisation is present.

12. *Pseudo-tuberculosis of Kidney. Sheep.* (287.)

The condition, which is a common one, is in no way related to true tuberculosis. The prescapular gland is most frequently affected, being filled with thick curdy contents. The disease is not known to affect man.

13. *Swine Fever. Ulcers of Intestine. Pig.* (69.)

The ulcers show swollen granulations projecting from their surface, probably part of the healing stage.

14. *Swine Fever. Heart,* showing numerous hæmorrhages in *Auricle and Ventricle. Pig.* (43.)

15. *Contagious Pleuro-pneumonia. Cow.* (28.)

Shows marbling of the lung, the white strands being the swollen fibrous tissue, the red the pneumonic lung tissue proper.

16. *Contagious Pleuro-pneumonia. Cow.* (73.)

Shows marbling of the lung, the white strands being the swollen fibrous tissue, the red the pneumonic lung tissue proper.

17. *After-effects of Contagious Pleuro-pneumonia. Cattle.* (282.)

Portions of lungs of two bullocks showing after-effects of pleuro-pneumonia contagiosa (?).

Microscopically sections show fibrosed septa and considerable proliferation of the epithelial cells of the pulmonary alveoli.

18. *Normal Lung. Cattle.* (82.)

Note the smooth surface of the healthy lung and the pink colour, as compared with the lungs in pleuro-pneumonia.

19. *Contagious mammitis. Cow.* (237.)

Teats and milk sinuses. Infected, yellowish; healthy, whitish.

20. *Putty-like Brisket. (Calcareous deposit.) Cattle.* (289.)

The deposit is crystalline, effervescing with acids. The cause of the condition, which is not rare, is not known.

20A. *Putty-like Brisket. (Calcareous deposit). Cattle.* (503.)

A putty-like mass, consisting of carbonates, &c., enclosed in connective tissue septa.

21. *Carcinoma of Bladder of Cow,* subsequent to endemic hæmaturia; also *Gland from Portal Fissure. Illawarra Redwater.* (547A.)

22. *Carcinoma of lumbar glands* (primary growth in bladder). *Cow.* Subsequent to endemic hæmaturia. (547B.)

An enormous carcinomatous growth occupies the bladder (A), and the lumbar glands (B). In the former, above the malignant mass may be seen the typical lesions of endemic hæmaturia. Compare with Nos. 23, 24.

23. *Bladder of Cow,* showing vascular growths. *Illawarra Redwater* (269A).

24. *Bladder of Cow,* showing vascular growths. *Illawarra Redwater* (269B).

A. Showing telangiectases and a papilloma.

B. Showing telangiectatic growths.

The cause of this condition in cattle is not known. It is possible that the condition may occur in man.

25. *Tumour. Heart. Bullock.* (39.)

Note the very large white mass with hæmorrhages attached to the heart.

The growth is sarcomatus and is invading the heart muscle towards the apex of the heart. Microscopically it consists of large cells in a fibrous matrix.

26. *Papilloma of Penis. Horse.* (649.)

27. *Squamous Epithelioma. Head of Rooster.* (279.)
Typical epithelial columns and cell-nests.

28. *Melanosis of Liver. Sheep.* (64.)

The interlacing black lines are caused by the deposit of melanin between the liver lobules. In this disease the portal glands are also pigmented. The condition is common in Australia. The livers though unsightly are not harmful to man.

29. *Generalised Melanosis. Young bullock.* (59.)

Note the extensive black areas, in the case of the lungs chiefly confined to the pleura, though with streaks into the lung tissue.

30. *Melanosis of Lungs. Bullock.* (204.)

31. *Pigmentation of Fat and Lymphatic Tissues. Pig.* (645.)

Apparently due to the deposit of a lipochrome. The pigs are believed to have been fed on penguins (*Eudyptula minor.*)

32. *Pigmentation of Omental Fat. Pig.* (645.)

See No. 31.

33. *Calculi* (triple phosphates). *Bladder. Bullock.* (53.)

34. *Specimens of Fat Dissociation.* Some treated with copper acetate (blue). An actinomycotic abscess is also present. *Cattle.* (306.)

This condition is common; its cause unknown.

35. *Fat dissociation round Kidney. Bullock.* (40.)

Kidney of young bullock showing fat dissociation, the so-called "Fat necrosis," a crystalline condition of the stearine in the fat cells surrounding the kidney.

36. *Gas-containing Vesicles attached to Mesentery. Pig.* (617.)

Small intestine. Gas consists chiefly of N with a little O. A not very rare condition.

37. *Necrosis of Muscle of Back,* probably due to injury. *Pig.* (643.)

38. *Tænia solium. Armed tapeworm of man.* (170.)

This tapeworm is very uncommon in Australia. It occurs in the intestine, and may reach a very considerable length. The larval form occurs in the pig.

39. *Tænia saginata. Tapeworm of man.* Sydney. (165.)

The unarmed human tapeworm. The larval form occurs as the bladder worm (*Cysticercus bovis*) in the muscles of the ox.

40. *Broad Tapeworm* (*Dibothriocephalus latus*). *Human.* (167.)

A human tapeworm which is very rare. The larval form occurs in certain fish.

41. *Roundworm from Man.* (*Ascaris lumbricoides.*) Sydney. (207.)

42. *Human Hookworm.* (*Ankylostoma duodenale*). (634.)

43. *Triocephalus trichiuris from cæcum of Orang Outan* (*Simia satyrus*), East Indies. Sydney Zoo. (531.)

Syn. *T. dispar.*

44. *Moniezia expansa. Tapeworm from Calf.* (208.)

45. *Moniezia planissima. Tapeworm from Cow.* Berry. (220.)

46. *Stomach flukes* (*Paramphistomum cervi ?*) *from Ox.* N.S.W. (348.)

47. *Worm-nests from Cattle. Onchocercu Gibsoni.* (All from one animal) Queensland. (460.)

One shows injury in surrounding tissue.

48. *Nodules in Intestine of Ox,* due to larval nematodes (*Œsophagostomum radiatum*). Richmond, N.S.W. (349.)

49. *Trichocephalus affinis. Cæcum of Ox.* N.S.W. (562.)

50. *Pentastomum denticulatum,* (*Linguatula serrata*). From mesenteric glands of cattle. Illawarra. (537.)

51. *Tick Fever. Hide, Liver, and Spleen. Cow.* (121.)

Numbers of ticks attached to hide. The females are large and the males much smaller, usually lying under cover of the female, with the rostellum attached close to it. This is the common cattle tick (*Margaropus annulatus.*)

52. *Tapeworm* (*Moniezia expansa*). *Sheep.* (163.)

Tapeworm from intestine of a sheep. Sometimes occurs in very great numbers in lambs, and then may produce death. (3 specimens.)

53. *Hæmonchus contortus. Stomach, Sheep.* West Australia. (428.)

54. *Dictyocaulus* (*Strongylus*) *filaria. Lung, Sheep.* N.S.W. (559.)

55. *Œsophagostomum columbianum. Intestine, Sheep.* N.S.W. (561.)

56. *Trichocephalus affinis. Cæcum. Sheep.* N.S.W. (560.)

57. *Larva of Sheep Bot, Œstrus ovis. Sheep.* N.S.W. (489.)

58. *Balbiania gigantea* (*Sarcocystis tenella*). *Sacs on Œsophagus. Sheep.* N.S.W. (302.)

59. *Tapeworm* (*Anoplocephala plicata*). *Horse.* (612.)

60. *Anoplocephala perfoliata. Intestine of Horse.* N.S.W. (381.)

61. *Ascaris megalocephala from Horse.* (392.)

62. *Spiroptera megastoma in Tumours in Stomach of Horse.* (146.)

Worm-nests in stomach of a horse, due to the presence of *Spiroptera megastoma.* These do not as a rule cause any serious trouble.

63. *Sclerostomum vulgare. Intestine, Horse.* West Australia. (445.)

64. *Oxyuris curvula. Horse.* N.S.W. (389.)

65. *Gastrophilus nasalis. Duodenum. Horse.* (218.)

66. *Bots Larvæ* (*Gastrophilus nasalis*). *Stomach of Horse.* (138.)
A common bot-fly larva from the stomach of a horse (N.S.W.).

67. *Bladder Worm* (*Cysticercus tenuicollis*) *on Liver of Pig.* Sydney. (367.)
This bladder worm, eaten by a dog, may give rise to a large tapeworm (*Tænia marginata*), sometimes over a yard long. To butchers, &c., this "bladder-worm" is known as a "water-ball."

68. *Ascaris suilla.* *Intestine of Pig.* West Australia. (187.)
The common round-worm from the intestine of the pig. This is closely related to, if not identical with, the common round-worm of man.

69. *Stephanurus dentatus.* *From Kidney of Pig.* Sydney. (579.)

70. *Strongylus apri* (*S. paradoxus*). *Lung of Pig.* (441.)

71. *Spiroptera strongylina.* *Stomach of Pig.* (564.)

72. *Gigantorhynchus gigas.* *Intestine of Pig.* (99.)
A hook-headed worm from a pig's intestine. It is not uncommon.

73. *Dipylidium caninum.* *Dog.* (135.)

74. *Ankylostoma caninum.* *Intestine of Dog.* (446.)

75. *Toxocara canis.* *Intestine, Dog.* (136.)
Round worm (*Toxocara canis*) from the intestine of a dog. This species also occurs in the intestine of cats.

76. *Tænia crassicollis.* *Intestine of Cat.* (134.)
The large tapeworm which infests the intestine of the cat. Its larval form is *Cysticercus fasciolaris*, which occurs in the liver of rats and mice.

77. *Cyst* (*Cœnurus serialis*) *in Limb of Rabbit.* (631.)

78. *Cœnurus serialis.* *Rabbit.* (168.)
This is from a rabbit, and is the bladder-worm stage of *Tænia serialis.*

79. *Coccidiosis or "Liver Rot" of Rabbit, due to Eimeria stiedæ* (*Coccidium oviforme*). N.S.W. (5.)

BOTANICAL SPECIMENS.

1. *Club-root of Cabbage.* *Plasmodiophora brassicæ.* (221.)
A protozoal parasite causing the growth of tumour-like masses on the roots.

2. *Lucerne*, showing *nodules*, due to Rhizobia. (45.)

3. *Dwarf Bean*, showing *root nodules*, due to Rhizobia. (50.)

4. *Cow Pea*, showing *root nodules*, due to Rhizobia. (47.)

5. *Parsnips.* (225.)
Root gall due to nematodes, *Heterodera radicicola.*

6. *Wheat.* (610.)
Flag smut, *Urocystis tritici.*

7. *Barley* affected with *Smut, Ustilago hordei; Rust, Puccinia simplex;* and *Leaf Streak.* (265.)

8. *Black rot* or *Bacteriosis* of Walnut. (457.)
Due to *Bacillus juglandis.*

9. *Grape rot.* (350.)
Due to *Gleosporium ampelophagum.*

10. *Apple Scab.* *Fusicladium dendriticum.* (475.)

11. *Celery leaf spot*, due to *Septoria petroselini.* (483.)

12. *Grey Scab of Orange*, due to *Sporodesmium griseum.* (499.)

13. *Tomatoes.* *Irish Blight* (*Phytophthora infestans*). (611.)

14. *Barley Smut* (*Ustilago nuda*). (295.)

15. *"Brown Fleck" or Internal Brown Spot in Potato Tubers.* (322.)

AUSTRALASIAN MEDICAL CONGRESS.

(NINTH SESSION.)

FIRST GENERAL MEETING.

HELD AT

THE GREAT HALL, SYDNEY UNIVERSITY.

MONDAY, 18TH SEPTEMBER, 1911, at 10 a.m.

Seated on the dais were:—The Honourable Sir Normand MacLaurin (Chancellor of the University of Sydney), Professor Allen, Hon. G. W. Butler, Dr. S. H. MacCulloch (Honorary Treasurer of Congress), Dr. G. A. Syme, Dr. Arthur Palmer (Honorary General Secretary of Congress).

Dr. F. ANTILL POCKLEY (President of Congress) in the Chair.

The PRESIDENT, who, on rising, was received with prolonged cheering, said:—

We have a report to be presented presently, which will be moved to be taken as read. As there is a great deal of matter to be considered, and as the Governor-General is holding a levee to-day at 12 o'clock, at which it is desired that many of us, if possible, should be present, it is desirable, without in any way suggesting that discussion on important matters should be limited, to request you to make your remarks as brief as possible. We are already a little late in starting, and our time is cramped.

The correspondence which is down on the business-paper as the first item we propose leaving until later. We will take first the Executive Committee's report, which I will ask the Honorary Secretary to move.

REPORT OF EXECUTIVE COMMITTEE.

Dr. PALMER: Mr. President, Ladies, and Gentlemen,—As you have in your hands a printed copy of the report of this committee, it is only necessary for me to move that it be taken as read and adopted.

Dr. S. H. MACCULLOCH seconded.

REPORT OF EXECUTIVE COMMITTEE.

At the final meeting of the Eighth Session of Congress, held in Melbourne, October, 1908, it was decided to hold the next meeting of Congress in Sydney in 1911, and Dr. F. Antill Pockley was elected President of the Ninth Session.

The President invited the Past President, Sir Philip Sydney Jones, and the President of the New South Wales Branch of the British Medical Association, to confer with him as to appointment of a General Committee. He also invited Dr. Arthur A. Palmer to act as Honorary General Secretary, and Dr. A. J. Brady as Associate Secretary, subject to confirmation by the General Committee.

As a result of the Conference various bodies were invited to nominate members for the General Committee.—

- The N.S.W. Branch of the B.M.A., 12 members in addition to the President.
- The various Suburban and Provincial Medical Associations, 1 member each.
- The Faculty of Medicine, 2 members.
- Medical Union, 3 members.
- Sydney University Medical Society, 1 member.
- The Royal Prince Alfred, Sydney, St. Vincent's Hospitals, 2 members each.
- Royal Alexandra, Royal North Shore, and Lewisham, 1 member each.

The Dean of the Faculty of Medicine, the Medical Heads of various Government Departments, Representatives of the Royal Army Medical Corps, the Medical Members of the Legislature, and the Secretary of the Medical Board were also invited to act on the Committee. Later the Committee thus formed added to its members by exercising its right of co-option.

The General Committee subsequently appointed an Executive Committee to carry out the arrangements for Congress. These Committees have held numerous meetings; the chief matters dealt with are referred to.

On 17th December, 1908, a meeting of the General Committee was held, when the appointments of Dr. Arthur Palmer as Honorary General Secretary, Dr. A. J. Brady as Associate Secretary, were confirmed, and Dr. S. H. MacCulloch was appointed Honorary Treasurer.

Standing Orders for the conduct of business were adopted. It was decided to get nominations from each State for Local Secretaries. A guarantee fund was established, each member's liability not to exceed £5.

The following resolution was carried:—"No medical man shall be accepted as a member of Congress except through the local Secretary for his own State or country, to whom his subscription must be paid."

At a meeting of the General Committee held on 18th June, 1909, the following resolution:—"That the General Committee of the Ninth Session of the Australasian Medical Congress in meeting assembled, desire to express their sympathy with the widow and family of the late Dr. John Thomson, former President of Congress, in their bereavement," was carried.

PATRONS.

Their Excellencies the Governor-General, the Naval Commander-in-Chief, the Governor of New South Wales, the Governors of New Zealand and of the various Australian States and of Fiji, the Lieutenant-Governor of New South Wales, the Lieutenant-Governor of New Guinea, and the Chancellor of the University were invited and consented to become Patrons of Congress.

In consequence of changes in office the Executive Committee later invited their Excellencies, the present Governor-General, Naval Commander-in-Chief, Governor of New South Wales, New Zealand, Victoria, and Fiji, to confer their patronage, and the Right Honorable the Lord Mayor of Sydney, was included.

The Premier and Government of New South Wales have accorded countenance and support to the Congress, and have promised to print the transactions of the session and to arrange various entertainments for the members during their stay in Sydney.

VICE-PRESIDENTS.

The former Presidents were appointed Vice-Presidents of Congress, together with the successive Fleet Surgeons, and the Director-General of the Army Medical Services.

J. C. Verco, M.D. (Lond.), F.R.C.S. (Eng.), President Adelaide Congress, 1887.

Sir Philip Sydney Jones, M.D. (Lond.), F.R.C.S. (Eng.), President Sydney Congress, 1892.

F. C. Batchelor, M.D. (Durh.), M.R.C.S. (Eng.), L.R.C.P. (Edin.), President Dunedin Congress, 1896.

Hon. G. H. Butler, M.R.C.S. (Eng.), L.R.C.P. (Lond.), President Hobart Congress, 1902.

Professor E. C. Stirling, C.M.G., M.A., M.D. (Cantb.), F.R.C.S. (Eng.), F.R.S., LL.D., President Adelaide Congress, 1905.

Professor H. B. Allen, M.D. (Melb.) (University of Melbourne), President Melbourne Congress, 1908.

Surgeon-General W. D. C. Williams, C.B., M.R.C.S. (Eng.), L.R.C.P. (Lond.), Director-General of the Army Medical Services of the Commonwealth, Melbourne.

Fleet-Surgeon V. Gunson Thorpe, M.R.C.S. (Eng.), L.S.A. (Lond.), Senior Naval Medical Officer, Australasian Station.

Fleet-Surgeon F. Falconer Hall, M.B., C.M., A.M., late Senior Naval Medical Officer, Australasian Station.

LOCAL SECRETARIES.

The following were appointed Local Secretaries :—

VICTORIA—Arthur Jeffreys Wood, M.D., B.S. (Melb.), 19 Collins-street, Melbourne.

QUEENSLAND—Wilton W. Love, M.B., C.M. (Edin.), Wickham Terrace, Brisbane.

SOUTH AUSTRALIA—John B. Gunson, M.B. (Adel.), M.R.C.S. (Eng.), North Terrace, Adelaide.

TASMANIA—Gregory Sprott, M.D., C.M., D.P.H. (Glas.), Macquarie-street, Hobart.

WEST AUSTRALIA—A. J. H. Saw, M.A., M.B., B.S., M.D., F.R.C.S. (Edin.), Perth, West Australia. *Acting Secretary*, W. Trethowan, M.B., M.S. (Aber.), Perth, West Australia.

NEW ZEALAND—South Island: Stanley Batchelor, F.R.C.S. (Eng.), L.R.C.P. (Lond.), Dunedin; North Island: W. E. Herbert, M.B., C.M., M.D. (Edin.), F.R.C.S. (Edin.), Boulcott-street, Wellington.

Dr. Saw was compelled to visit England, and Dr. Trethowan, of Perth, was appointed to fill the vacancy during his absence.

REPRESENTATIVES.

The following were appointed as the representatives of the various States, Capital Cities, and Associations :—

Government of Victoria—Dr. B. B. Ham.
Government of Tasmania—The Hon. G. H. Butler.
Government of West Australia—Dr. James Hope.
Government of Queensland—Dr. J. S. C. Elkington.
Government of South Australia—Dr. A. A. Lendon.
Government of New Zealand—Dr. T. H. A. Valintine.
Health Department of Tasmania—Dr. J. S. Purdy.
City of Hobart—Dr. Gregory Sprott.
City of Perth—Dr. Pope Seed.
City of Melbourne—Dr. Jamieson.
University of London—Dr. Maudsley, of Melbourne.
University of Oxford—Dr. E. J. Jenkins, Sydney.
University of Manchester—Professor J. T. Wilson.
British Medical Association—
Dr. Albert E. Martin, W.A.
Dr. J. R. Purdy, N.Z.
Dr. H. L. Maitland, N.S.W.
Dr. H. H. Fetherston, Vic.
F. S. Horne, S.A.

Twenty-seven honorary members have been elected, principally from learned Societies and University Teaching Staff.

It was decided to divide the Congress into twelve sections :—(1) Medicine, (2) Surgery, (3) Obstetrics and Gynæcology, (4) Anatomy, Physiology and Pharmacology, (5) Pathology, Bacteriology and Tropical Medicine, (6) Public Health and State Medicine, (7) Ophthalmology, (8) Psychological Medicine and Neurology, (9) Diseases of Children, (10) Naval and Military Medicine and Surgery, (11) Dermatology, Radiology, and Medical Electricity, (12) Laryngology, Rhinology, and Otology.

Invitations were forwarded to the various States and New Zealand, asking for nominations for the positions of Presidents and Vice-Presidents of Sections, and a Committee was appointed to select the officers and Committees for the various Sections for the consideration of the General Committee.

The Secretaries of Sections have had a great deal of work in getting together the programme of work which is submitted for the consideration of the Members, and they deserve hearty thanks for the way in which they have performed their duties.

It was decided that the only special address to be given in full Congress be that on Medicine.

Special Committees were appointed to report on Syphilis and Contract Medical Practice. These Committees have held a number of meetings, and the reports will be laid before Congress. The thanks of the Executive Committee are due to the members of these Committees for the excellent work done by them.

Dr. W. Moore, of Melbourne, resigned his position as convener of the Committee to report on Hospital Abuse, and Dr. Ralph Worrall was appointed to fill the vacancy.

The date of Congress was fixed for the 18th to 23rd September, and the subscription was fixed at £2 2s. for metropolitan members and £1 1s. for all other members.

An invitation was forwarded to the British Government, asking if they would send a representative. Sir Frederick Treves was appointed, but, to the great regret of the Committee, he was afterwards compelled to relinquish the appointment on account of ill-health.

At a meeting of the General Committee on 25th April, 1910, an Executive Committee was appointed to carry out all arrangements in connection with the Congress. This Committee from time to time exercised its right of addition, and now comprises:—

EXECUTIVE COMMITTEE.

President
Past President
General Secretary
Associate Secretary
Treasurer
Dr. G. H. Abbott
Dr. Geo. Armstrong
Dr. W. G. Armstrong
Dr. Richard Arthur, M.L.A.
Dr. E. V. Barling
Dr. Fourness Barrington
Dr. H. G. Chapman
Dr. C. B. Clubbe
Dr. Cyril Corlette
Dr. W. H. Crago
Dr. Gordon Craig
Dr. Andrew Davidson
Dr. J. A. Dick
Dr. T. Fiaschi
Dr. J. Foreman
Dr. J. Macdonald Gill
Dr. Sinclair Gillies
Sir Jas. Graham
Dr. J. C. Halliday
Dr. Herschel Harris
Dr. H. C. Hinder
Dr. J. L. T. Isbister
Dr. R. H. Jones
Dr. C. H. E. Lawes
Dr. W. F. Litchfield
Dr. Charles MacLaurin
Dr. H. L. Maitland
Dr. Odillo Maher
Dr. H. H. Marshall
Dr. A. E. Mills
Dr. W. McMurray
Hon. Dr. J. B. Nash, M.L.C.
Dr. B. J. Newmarch
Dr. Russell Nolan
Dr. R. Paton
Dr. Cecil Purser
Dr. Clarence Read
Dr. G. E. Rennie
Dr. Reuter E. Roth
Dr. Eric Sinclair
Dr. R. Scot Skirving
Dr. S. A. Smith
Dr. E. Stokes
Prof. Anderson Stuart
Dr. Ashburton Thompson
Dr. R. H. Todd
Dr. R. B. Wade
Prof. Welsh
Prof. Wilson
Dr. P. M. Wood
Dr. Ralph Worrall
Dr. H. C. Taylor Young

A Reception and Entertainment Committee was appointed as follows:—

Reception and Entertainment Committee.

President
General Secretary
Treasurer
Dr. A. J. Aspinall
Dr. A. J. Brady, Hon. Sec.
Dr. J. Foreman
Dr. J. Macdonald Gill
Dr. Sinclair Gillies
Dr. E. J. Jenkins
Dr. R. H. Jones
Dr. E. Sydney Littlejohn
Hon. Dr. Mackellar, M.L.C.
Dr. H. J. Marks
Hon. Dr. J. B. Nash, M.L.C.
Dr. R. Pope
Dr. Cecil Purser
Dr. Clarence Read
Prof. Anderson Stuart
Dr. D. Thomas
Dr. E. T. Thring
Prof. Welsh
Prof. Wilson
Dr. Taylor Young
Dr. F. S. W. Zlotkowski

The following were appointed the

Literary Committee.

President
General Secretary
Treasurer
Dr. G. H. Abbott
Dr. Fourness Barrington
Dr. W. F. Litchfield
Dr. G. E. Rennie
Dr. R. Scot Skirving

With the assistance of the Associate Secretary of Congress, and the Secretaries of Sections.

The following were appointed the

Congress Museum Committee.

President
General Secretary
Hon. Treasurer
Professor Wilson
Professor Welsh
Dr. G. H. Chapman
Dr. Fourness Barrington, Hon. Sec.
Dr. Taylor Young
Dr. Sydney Jamieson
Dr. W. H. Crago

with power to add to their number.

A resolution of condolence was forwarded to Miss Rawson on the death of her father, the late Sir Harry Rawson, one of the Patrons of Congress.

The Federal Government was asked to bear the expense of bringing six filarial patients from Queensland to the Congress as exhibits. The Government has kindly consented to do this.

The Honorary Treasurer received the sum of £378 5s. 3d. from the Honorary Treasurer of the Eighth Session of Congress (Dr. G. A. Syme).

At the Executive meeting of 10th May, 1910, the following resolution with reference to the death of His Majesty the King was carried:—

"The President and Executive Committee of the Ninth Session of the Australasian Medical Congress, representing the medical profession of Australasia, desire to convey to Her Majesty the Queen Mother Alexandra and the Members of the Royal Family their sincere sympathy on the occasion of the death of His late Majesty King Edward VII. The late King having been an Honorary Fellow of the Royal College of Physicians of London and of the Royal College of Surgeons of England, they desire especially to express their sense of the great loss of so distinguished an ornament of the profession."

His Excellency Sir William Macgregor was invited to become President of the Section of Pathology, but he found that it would not be possible for him to be present at Congress.

A Ladies' Committee has been formed, with Mrs. Antill Pockley as President; Mrs. W. G. Armstrong, Honorary Secretary; Mrs. Steer Bowker, Honorary Treasurer. The Executive would express their thanks to the Ladies' Committee for the excellent organising work done, and for the entertainments they have offered.

There are 922 members enrolled—from Victoria 208, from South Australia 61, from New Zealand 34, from Queensland 51, from Tasmania 28, from West Australia 19, from New South Wales 521.

The report of the Special Committee, appointed to deal with the question of Contract Practice, has been approved by the Executive, and will be submitted for adoption separately.

Somewhat germane to the matters therein considered is one that has come into prominence since last meeting of Congress—one that has taken shape in Great Britain in the National Insurance and Invalidity Bill, and, under the name of nationalisation of hospitals and medical services generally, is threatening us here.

The Executive would suggest the advisability of appointment by Congress of a Special Committee of representatives of all the States and of New Zealand to formulate in anticipation a concerted plan of action, so that in the event of legislation being introduced here we should be ready for it. Only by being prepared beforehand has the profession in Great Britain been able to so successfully compel recognition of its claims by the Chancellor of the Exchequer. The Committee might comprise the Presidents for the time being of the various branches of the British Medical Association, and Presidents and past Presidents of Congress, with power to add to their number, as one likely to command the confidence of the profession. The funds at credit of Congress might be put to a worse use than paying necessary expenses of printing, travelling, &c.

Another question that might be discussed is the desirability of forming a Standing Committee to arrange for Congresses, such as they have in Canada. All that Congress does now is to decide on place and approximate date of meeting of the succeeding session, and to elect a president. That places the initial duty and responsibility of appointment of committees and officers of sections on the President, and there is no provision for appointment of a successor should the President, in the interval between sessions, die or resign, or be incapacitated from acting.

F. ANTILL POCKLEY,
President.

ARTHUR PALMER,
Honorary General Secretary.

The motion was put to the meeting and, without discussion, carried unanimously.

The PRESIDENT: Professor Allen wishes to move a resolution which I feel sure will appeal to all of us.

Professor ALLEN: I have to move, "That the Australasian Medical Congress, now assembled in Sydney, place on record its profound regret at the death of Colonel John Thomson, President of the Fifth Session, and its appreciation of his lofty character and his eminent services to the public and the profession, and that the President be requested to transmit this resolution to Mrs. Thomson."

Dr. BUTLER: I wish to second that.

The motion was carried in silence, all members standing in their places.

FINANCIAL.

The PRESIDENT: I will now ask the Treasurer of last Congress to move the adoption of the financial statement, which you will see printed at the end of the Executive Committee's report.

Dr. G. A. SYME (Honorary Treasurer of Eighth Session of Congress): I have very much pleasure in moving that the statement of receipts and expenditure, as printed, be taken as read and adopted.

Dr. MACCULLOCH: I second that.

AUSTRALASIAN MEDICAL CONGRESS, EIGHTH SESSION, MELBOURNE, 1908.

STATEMENT of Receipts and Expenditure.

Receipts.	£	s.	d.	*Expenditure.*	£	s.	d.
Remittances from 7th Session, Adelaide, 1905 ...	295	0	0	General Expenses of Congress	455	19	9
Members' Subscriptions ...	829	16	4	Printing, Stationery, &c...	177	12	0
Grant from Commonwealth Government	56	4	0	Transactions	207	9	11
Interest	21	5	8	Bank Charges and Exchanges	6	13	9
Sundry Receipts, Refunds, Exchanges paid by Members, &c.	25	16	8	Audit Fee	2	2	0
				Remitted to 9th Session, Sydney, March, 1909...	300	0	0
				Balance, to be remitted to 9th Session, Sydney, March, 1911	78	5	3
	£1,228	2	8		£1,228	2	8

Examined and found correct.
J. V. M. WOOD, F.C.P.A.,
Incorporated Accountant.

G. A. SYME,
Hon. Treasurer.

Melbourne, 23rd March, 1911.

STANDING ORDERS.

President: Dr. Palmer wishes to move an addition to the Standing Orders of Congress.

Dr. Palmer: The alteration of the Standing Orders, which I have to ask the meeting to adopt, is only a slight one in the first section, which at present reads:—" Ordinary members of Congress shall be medical practitioners, legally qualified to practise in their respective countries, who have paid their subscriptions to the Treasurer of Congress or to the corresponding State Secretary." Owing to certain friction having arisen on previous occasions, the Executive of this Congress, at its first meeting, carried a resolution that nobody would be accepted as a member of Congress except through his own State Secretary, as it is very often quite impossible to know anything at all about medical men from another State or New Zealand.

I, therefore, wish to move that this first section be so altered that practitioners shall be eligible for membership after paying their subscriptions to the Secretary of their own State. Each man must join in his own State.

Professor Allen: I beg to second that motion.

President: The resolution is that in the first section of the Standing Orders the words " or to the " be struck out, and the word " through " substituted. The section will then read:—

" Ordinary members of Congress shall be medical practitioners legally qualified to practise in their respective countries, who have paid their subscriptions to the Treasurer of Congress through the corresponding State Secretary."

Carried unanimously.

STANDING ORDERS.

1. Ordinary Members of Congress shall be medical practitioners legally qualified to practise in their respective countries, who have paid their subscriptions to the Treasurer of Congress through the Corresponding State Secretary.

2. The Executive Committee reserve the right to refuse or to return any subscription.

3. The subscription shall be determined for each Session by the Executive Committee thereof, and shall be payable to the Treasurer or State Secretary, and shall entitle the member to a copy of the published Transactions of the Congress, if any.

4. Honorary members, not necessarily legally qualified medical practitioners, may be elected by the Executive Committee.

5. There shall be a Preliminary General Business Meeting and an Inaugural General Meeting of Congress on the first day, and a Final General Business Meeting on the last day of the Session. Other General Meetings may be held in pursuance of resolution of the Congress or of the Executive Committee.

6. At the Preliminary General Business Meeting, the report of the Executive Committee shall be submitted, and also the report of the Treasurer of the preceding Session. Other business shall be as arranged by the Executive Committee. Resolutions may be submitted by members of Congress, but only after notice of motion, which must be in the hands of the General Secretary at least a week before the first day of the Session.

7. The Inaugural General Meeting shall be devoted entirely to the usual addresses and resolutions. The only speakers shall be those designated by the Executive Committee.

8. At the Final General Business Meeting, the place and date of the next Session of Congress shall be determined, and the President for that Session elected. Resolutions carried in any Section or Sections and ordered by any Section to be reported

to the Congress shall be considered. Other resolutions carried in Section shall not be reported. Motions may be submitted on behalf of the Executive Committee. Other motions, of which at least twenty-four hours' notice has been given, may be submitted. The President shall have full power to restrict debate.

9. Subject to any resolutions of Congress, the business at other General Meetings shall be arranged by the Executive Committee, which may, as far as it deems desirable, designate the readers of papers and the speakers.

10. The proceedings of each Section shall be regulated by the President of the Section at his discretion, provided that a provisional programme of work for each Section may be published by the Executive Committee after consultation with the Sectional Officers. Two or more Sections may meet together for any joint work.

11. The Executive Committee shall determine which addresses, if any, by Presidents of Section shall be given in full Congress and which shall be given in the Section. With the exception of Addresses to be given in full Congress, the Address in any Section may at the discretion of the President of Section be replaced by a paper or by the opening of a discussion by the President of Section.

12. The President of the Congress shall preside at all General Meetings of the Congress, unless the Executive Committee shall with his consent have arranged otherwise. In the absence of the President, the Past President of longest standing present shall preside. If this provision fails, the meeting shall elect a Chairman.

13. In Sectional Meetings, in the absence of the President of the Section the available Vice-President whose name is highest in the list of Vice-Presidents shall take the Chair. If this provision fails, the Section shall elect a Chairman.

14. Any debate in General or Sectional Meeting may be interrupted by motion proposed and seconded that the question be now put, or that the meeting pass on to the next business. The motion in either case shall be put without further debate, and, if it is carried, the question shall be put, or the next business called on, as the case may be. Otherwise the decision of the President or Chairman in the conduct of any discussion shall be final.

15. Unless expressly provided otherwise by the Executive Committee no paper nor report may occupy more than twenty minutes. No speaker in any discussion may occupy more than ten minutes. The author of any paper or report shall have the right of reply not exceeding five minutes.

16. Notice of all papers, exhibits, &c., to be submitted should be in the hands of the General Secretary or Secretary of Section two months before the opening of Congress, otherwise they will probably not be included in the published programme of Sectional work. Papers may, however, be accepted up to and even during the meeting of Congress. All notices of papers, exhibits, &c., are subject to acceptance by the Executive Committee, or in emergency by the President of the Section concerned.

17. Reports prepared at the request of the Executive Committee or of any Sectional Committee should be in the hands of the General Secretary or Secretary of Section at least one month before the opening of Congress, and as far as possible such Reports will be printed for distribution at or before the Congress.

18. Short abstracts of papers should be forwarded to the General Secretary or Secretary of Section one month before the opening of Congress, and as far as possible such abstracts will be printed for distribution at or before the Congress.

19. Members detained in other Sections may forward to the Secretary of any Section a brief note to be incorporated in any discussion or in the report thereof. Such note must reach the Secretary before the close of the discussion in question.

20. All reports or papers submitted to any meeting of Congress shall be handed to the General Secretary or the Secretary of Section before the close of that meeting. They may not be subsequently altered without the consent of the Executive Committee.

21. No vote of thanks shall be proposed to the author of any paper.

22. The Executive Committee reserves the right to determine for itself, or by any Committee appointed for the purpose, what reports, papers, or speeches, or what parts of reports, papers, or speeches shall be published in the Transactions of the Congress.

Carried unanimously.

CORRESPONDENCE.

President: There are one or two letters which the Honorary General Secretary will now read.

Dr. **Palmer**: The first is from Dr. Douglas Mawson, who, under date 11th September, writes:—" Dear Sir—There is a dearth of applications for positions on the staff of this expedition "—that is, the Australasian Antarctic Expedition—" by qualified medical men. I feel sure that this is due to lack of public knowledge regarding our plans, and ask you to take the opportunity of the Medical Congress to let it be known that several medical posts are still vacant. About three are wanted for the land parties, and one for the ship. Applicants should be young, strong, and vigorous. It will be greatly to the advantage of the expedition should the medical officers have a knowledge of kindred sciences, that is, biology. Their services will be required from November 25th, 1911, to April 30th, 1913. All expenses are paid and clothing equipment found; and, further, a sum of £300 will be paid to each on return to Australia." (Laughter.) " I must apologise for troubling you with this matter; but it should be a good opportunity for medical men interested in scientific research."

There is another letter, from the Commonwealth of Australia, Department of Trade and Customs:—" I beg to inform you that Dr. J. H. L. Cumpston, Acting Director of Quarantine, will attend as the official representative of the Commonwealth at the Australasian Medical Congress to be held in Sydney during the current month."

CONTRACT PRACTICE COMMITTEE.

Dr. **Charles Lawes**: Mr. President, Ladies and Gentlemen,—I have much pleasure in proposing the adoption of the report of the Contract Practice Committee. It is printed, and I move that it be taken as read and adopted. In doing so, I should like to emphasise a few points in this report, very briefly. The first is that the income limit shall be enforced, as far as possible, in each State. The South Australian Defence Association suggested that the time was not yet ripe for them to take any action in this matter—not until there was simultaneous action throughout the various States. The success of the profession has been so satisfactory in New South Wales that I do not think there would be in tackling this question any danger of men coming from other States to take up positions.

Another matter which the Committee thought important was the state of the hospitals in West Australia; but, as the Hospital Committee's report deals fully with that, I need not touch upon it.

The next thing is the question of lodges of which members of this Medical Association would not accept the officership, and the principle the Committee felt they wanted to lay down was that medical men should be paid for the work they do; that is to say, they should be paid a fee of so much per patient, and not accept a lump sum regardless of the number of persons enrolled on the honorary lodge lists. We felt that the establishment of an indemnity fund, out of which medical officers could be refunded for any losses they sustained through following out this work, would be in the best interests of the profession.

The last matter is as to universal military training; but, as Dr. Thomas has a motion on the table in that connection, I will not deal with it.

I have very much pleasure in proposing that the report, as printed, be adopted.

REPORT OF CONTRACT PRACTICE COMMITTEE.

In conformity with the resolution passed at the Eighth Session of Congress, a Contract Practice Committee was appointed and now begs to submit the following report:—

At the Eighth Session of the Australasian Medical Congress, held in Melbourne in October, 1908, the following resolution was passed:—"That no medical man should pass for admission into any friendly society, lodge, club, or association, or private list, for professional attendance at contract rates of remuneration, any person whose income from all sources, together with that of his wife (if any), exceeds £4 per week, that is, £208 per annum. Further, no medical man should attend professionally at contract rates any member of any of the above-mentioned organisations, whose income from all sources, together with that of his wife (if any), exceeds the sum of £6 per week, that is, £312 per annum."

One of the first actions of this Committee was to inquire into what steps had been taken in the different States to bring the above resolutions into force. It was found that New South Wales was the only State that had definitely taken steps to do this, and the efforts of medical men in this State have been very successful. The result has been that these wage-limit clauses have been already introduced into agreements between medical officers and their lodges, not only in the city but also in the country, in most of the more populous parts of New South Wales, and it is estimated that some 500 practitioners in this State, who are medical officers of lodges, have this clause embodied in their agreements with their lodges.

In Victoria nothing had been done until quite recently, as it was recognised that the first step to be taken was to enlarge the membership of the Victorian Branch of the British Medical Association. In May of this year, however, the Victorian Branch of the British Medical Association adopted a resolution in regard to the proposed Friendly Societies' Hospital to the effect that before the Association could in any way co-operate, it was essential that the Friendly Societies should adopt an income limit in accordance with the resolution of the 1908 Congress.

In Western Australia, New Zealand, South Australia, and Tasmania no steps have been taken in the matter.

The Secretary of the Defence Association of South Australia, in writing to the Committee on this subject, stated that its members, though approving the principle, felt that it was impossible to carry it out in South Australia, unless it were simultaneously carried out throughout Australasia.

This Committee feels that the success of the efforts of the profession in New South Wales show that it is possible for each State to work out its own salvation.

In Queensland the wage-limit clause has, so far, not been enforced, but the Queensland Branch of the British Medical Association is at present taking steps to draw up a Model Lodge Agreement with this clause included.

This Committee wishes to strongly urge the Medical Profession of Australasia to make a determined stand to enforce these wage-limit clauses in all lodge agreements, as it feels that it is a matter of vital interest to all lodge practitioners.

Lodge Rates in the Different States.

The following are the average rates paid by lodges, so far as could be ascertained in the different States:—

	N.S.W.	Victoria.	S. Aust.	Queensl'd.	Tasmania.	W. Aust.	N. Zealand.
City and Suburban.	16/- to 20/-	14/- to 16/-	25/- to 30/- with medicine.	15/- to 18/-	12/- to 15/-	15/- to 16/- 20/- to 22/6 with medicine.	16/-
Country ...	26/- to 52/- with medicine.	16/- to 20/- up to 30/- with medicine.	30/- to 60/- with medicine.	18/- to 25/-	30/- with medicine.	26/- with medicine.	22/6

The above rates are for member and family, and only include medicine when so stated.

MIDWIFERY, OPERATIONS AND ANÆSTHETICS.

New South Wales.—Midwifery, 21s. to 42s. Operations and anæsthetics, a matter for arrangement between member and medical officer. Almost invariably extra.

Victoria.—Midwifery cases extra, and as a rule no concession to lodge patients. Operations and anæsthetics not included, except in A.N.A. agreement.

Western Australia.—All extra.

Queensland.—Midwifery: Town, 21s. to 42s.; Country, 42s. to 63s. Operations and anæsthetics extra.

New Zealand.—All extra.

South Australia.—All extra.

Tasmania.—All extra.

CONTRACT PRACTICE AT MINES.

The arrangements and rates in the different States are as follows:—

New South Wales.—There is a contract usually between the Medical Officer and a "Medical Committee" of the miners themselves. Married men pay from 1s. to 1s. 6d. per fortnight, and boys under 16, 6d. to 1s. per fortnight. This is usually deducted from their wages at the Mine Office. These rates include medicine.

Victoria.—At Wonthaggi (the State Coal Mine) a State Medical Officer is partly paid by weekly sums deducted from the wages of the employees. Information as to other mines was not procurable.

West Australia.—At most mining centres there is a "Local Committee Hospital," to the funds of which single men pay 1s., married men 1s. 6d. per fortnight. The Medical Officer to the Hospital at a fixed salary has to attend all subscribers without other remuneration. Where there is a Government Hospital miners pay 1s. direct to some medical man, who provides hospital accommodation, medicines, and attendance.

Queensland.—At Mount Morgan a similar state of affairs to that existing in West Australia obtains. All employees who are not in lodges pay 16s. per annum to the local hospital, and so become entitled to attendance by the Medical Officer to the Hospital. At Gympie there is not a contract with the mines, most of the men being members of lodges.

New Zealand.—Medical Officers paid by the miners, a certain amount being deducted from their wages at each pay.

South Australia.—At Moonta single men under 18 years pay 13s. per annum, and 18 years 26s. per annum. Married men, 52s. per annum.

Tasmania.—Medical Officer paid by men by means of fortnightly deduction from their wages.

Country Hospitals.

The Committee made inquiries concerning conditions of the contracts between Medical Officers and Hospitals in country towns.

New South Wales.—In only two prominent hospitals are there resident Medical Officers—namely, Newcastle and Goulburn, the former having two and the latter one. These officers are paid a fixed salary, and are not allowed private practice. In all other country towns the Medical Officers are visiting ones. In some all are honorary, in others one may be paid and the rest honoraries, or in others more than one may be paid.

Victoria.—No definite information to hand.

Western Australia.—Country hospitals are of two kinds—(*a*) Government Hospitals, (*b*) Local Committee Hospitals. (*a*) At Government Hospitals Medical Officers get a fixed salary for all *bonâ fide* hospital work and all Government work; (*b*) At Committee Hospitals (subsidised by Government) the Medical Officer has to attend to all hospital patients and all subscribers, even at their own houses, free. However, in some hospitals, anæsthetics, confinements, and operations are extra, while, again, in other hospitals these are not extras. The Medical Officer gets a fixed salary with right of private practice. As these local committees never refuse any subscriptions, there is necessarily very little private practice. At some of these hospitals residence is provided for Medical Officer, at others it is not provided.

Queensland.—Resident: Usually a paid Medical Officer, with right of consultation practice, when he is a senior man, salary £300 to £500 and quarters. Or a junior man, devoting his whole time to the hospital, at £200 a year, working entirely under the Honoraries. Non-resident: Visiting Medical Officers at fixed salary, with full right of private practice.

South Australia.—Non-resident Medical Officer at a salary, with right of private practice.

New Zealand.—Medical Officer paid, either resident or non-resident, with right of private practice. The paid resident, with right of private practice, is considered very unsatisfactory.

Tasmania.—There are no country hospitals paying a Resident Medical Officer. A Hospital Union maintains the Hospital, and the Medical Officers of the Lodges and Union treat suitable cases there.

CONTRACT WORK FOR GOVERNMENT INSTITUTIONS, SUCH AS STATE SCHOOLS, RAILWAYS, PRISONS, &c.

This Committee made inquiries on this subject, and elicited the following facts. In this connection whole-time appointments are not included, only those where a medical man in general practice agrees for a certain sum to do work for the institutions mentioned.

In Queensland Railways, one man in Brisbane has a salary of £150 per annum for examining new applicants only. This works out at about 5s. per examination. In addition to this he gets all other work in connection with Railways in Brisbane and district, for which he charges full private fees, paid by the Railway Department.

In New South Wales Education Department there is a Medical Adviser, whose salary is £560 per annum, with right of private practice, and three women Medical Inspectors of school children, one of whom is in receipt of a salary of £400, with right of private practice; another, who receives £250 per annum, with right of private practice; while the third is paid by fees.

In many of the country towns of New South Wales the Gaols are attended by local medical practitioners, who receive from £40 to £102 per annum. In New Zealand the Police, Prison, and Permanent Artillery Force each have a special Medical Officer, who is a general practitioner, and who is paid about £100 per annum. The Railways retain a special man, to whom all medical matters are referred, but who is paid for each item, and not by salary.

In Hobart a lot of the Government work is done by one man, who is paid £150 per annum. His duties are:—Attendance on the sick poor at their houses, attendance on the inmates of the charitable institutions of New Town, consulting Medical Officer to the Railway Department and to the Attorney-General's Department. The same man acts as Medical Officer to the State Schools in Hobart for a salary of £50 per annum.

LODGES AND BENEFIT SOCIETIES, OF WHICH MEMBERS OF THE DIFFERENT BRANCHES OF THE B.M.A. WILL NOT ACCEPT THE MEDICAL OFFICERSHIP.

It was found there were several of these in the different States. The reasons why medical men will not act for these lodges are as follows:—

In some cases the lodge or society will not pay its medical officers the rate per member fixed by the local Medical Association or by the medical officers practising in the district. In others the lodge pays its medical officers by salary instead of at a certain rate per member per annum, and the salary is inadequate. In one such institute in Queensland the salary at one time worked out at about 7s. per member and family per annum. In another well-known case a certain association is regarded by the profession in Queensland and New South Wales not a *bonâ fide* friendly society, but a political organisation, with medical benefits thrown in to attract members to join.

One benefit society in New South Wales is a proprietary institution, with paid canvassers.

Private Lists are in vogue in a few towns in the different States, and a resolution has been carried by the New South Wales Branch of the British Medical Association discountenancing them, and the Committee supports this resolution.

Indemnity Fund.—It was found that in only one State, namely New South Wales, has any provision been made for a fund to indemnify practitioners who have lost their lodges through loyalty to the rules and resolutions of the British Medical Association, and even in New South Wales the fund exists really as a nucleus which can be easily increased if occasion arises. The Committee is of opinion that if such a fund existed in all the States it would greatly facilitate the bringing about of reforms in contract practice.

MEDICAL APPOINTMENTS IN THE PUBLIC SERVICE.

The question has arisen in the course of inquiries made by the Committee as to whether medical appointments to the Public Service, Federal or State, should be made without applications being first invited by advertisements.

MEDICAL OFFICERS UNDER THE UNIVERSAL MILITARY TRAINING SYSTEM.

The matter of the remuneration of medical men who accept the position of Area Medical Officers has been carefully considered by the Committee. The present rate of remuneration works out at about 1s. per examination, and in large areas even as low as 9d. per examination. This is held by the Committee to be quite inadequate, and it suggests to Congress that the amount paid for each examination should be materially increased.

G. H. ABBOTT, Chairman,
C. H. E. LAWES, Secretary,
Contract Practice Committee.

Dr. G. A. SYME: I second that.

Dr. E. W. MORRIS (South Australia): The rates for city and suburbs in New South Wales and Victoria are flat rates, chargeable to each member; but you would put us down in South Australia as 25s. to 30s. with medicine. We have not got a flat rate. About five-sixths of the members in South Australia are single members, paying only 10s. with medicines, and there is a very large proportion of juveniles there who only pay 6s. I might also state, on behalf of the Medical Defence Association in South Australia, we think there is not the slightest doubt that an indemnity fund will be started this year.

I might say, personally, that I am very glad to see that such a prominent position is to be given to the question of the payment of area officers and naval and military officers generally under the new defence scheme.

Dr. G. A. SYME: I move, as an amendment, that some more absolutely definite resolution of the Committee be adopted. There is no summary of actual recommendations to be adopted by the Congress. I move that the report be sent back to the Committee with the suggestion that a schedule of the recommendations to be adopted by this Congress be submitted. I understand that the Committee has really prepared the recommendations; but it did not consider it was within its function to submit them to the Congress.

Dr. CROWTHER (Tasmania): I second that. I think time should be given. I am agreeable to a lot of it, but there is a great deal of it I would not care to express an opinion about until I have had an opportunity of giving it careful consideration. I have been attached to friendly societies for over thirty years, and I do think further action on this matter should be reserved.

Dr. Worrall: This report is a very serious matter for everyone connected with medical societies, and if it is not dealt with now, there will be very little opportunity of discussing or endorsing it later. It has been gone into very fully, and it deals with broad and general principles, which, if adhered to, can be so arranged in each State as the local exigencies of the profession demand. I trust that the Congress will carry it by a large majority, or unanimously.

President: The Committee had various recommendations in certain parts of the report; but, as they were asked merely to report, they felt that it was not within their province to make recommendations; they felt that they should do merely what they were asked to do. This is a statement of fact, and I do not know that anything could be gained by considering facts; but would it do—I only make it as a suggestion—if recommendations, based on this report, were brought up at Saturday morning's meeting?

A Member of Congress: A good idea.

President: Then you will have time to consider it in detail, and it will not reflect in any way on this Committee. We purposely left out controversial matters, which matters of opinion would necessarily be, and confined the report, as far as we could, to statements of fact. These are the conditions regulating contract practice, as far as we have been able to find out, in the different States, and if this report were adopted, it would not in any way prevent recommendations being made on Saturday morning. Indeed, we would welcome them. That is what the object of the report is—to place you in possession of the facts so that you may be able to make recommendations. (Hear, hear.)

Dr. G. A. Syme: If someone will move that an opportunity be given to discuss recommendations, I will withdraw the amendment.

President: The amendment having been withdrawn with the consent of the meeting——

Dr. Crowther (Tasmania): Pardon me! I have not consented. This has just been placed in our hands. We have not had time to read it. It is an important matter. I have just had this report placed in my hands. It says that no lodge should be taken under certain conditions. I am not agreeable to that, because I know there are a large number of poor people who have a very hard struggle to scrape together their lodge contributions. I ask for time to carefully look through this report. That is all I want. So far as my individual opinion goes, I do not want to support it without having time to consider. That is all I want to say.

President: I should like to point out that what Dr. Crowther is objecting to is not a recommendation of the Contract Practice Committee at all; it is a resolution adopted by last Congress, and it has nothing to do with this report.

The motion having been put to the meeting, it was agreed unanimously that the report of the Contract Practice Committee be adopted.

Dr. Lockhart Gibson moved, Dr. Nash seconded, that an opportunity be given at the meeting on Saturday morning for the discussion of recommendations on the report.

President: The recommendations will be submitted to Saturday morning's meeting; they will be prepared and circulated as early as possible before that, in order that you may have time to consider them.

HOSPITAL ABUSES.

Dr. WORRALL: Mr. President, Ladies, and Gentlemen—At last Congress the following resolutions were unanimously carried, with one dissentient, who voted under a misapprehension and who is now entirely in accord with them:—"(1) That in the opinion of this Congress no payment whatever should be taken from patients treated at hospitals maintained by the donations of the charitable and by Government grants. (2) That, excepting cases of accident or emergency, every patient should, before being treated, be required to sign a declaration of inability to pay for medical attendance, and that, as far as possible, all patients seeking treatment should bring a recommendation from their own medical attendant, whether lodge or other. (3) That this Congress affirms the foregoing resolutions as the foundation of any real reform in the present abuse of hospitals, and is of opinion that other hospital accommodation would extend naturally to the inclusion of all those able to pay for medical or surgical treatment. (4) That this Congress is of opinion that it would be a great gain to the community if the executive body were established to,—(*a*) Administer the Government grant, (*b*) classify hospitals, (*c*) prevent overlapping of hospital work, (*d*) insist on inquiry for the prevention of imposition, (*e*) insist on the adoption of uniform methods of treating accounts, (*f*) close unnecessary hospitals, (*g*) prevent the establishment of unnecessary hospitals, (*h*) foster economy in hospital administration, (*i*) promote the transfer of patients from one class of hospital to another."

In the *Daily Telegraph* of last Saturday, Mr. President, there was a pronouncement of one of the Ministers of this State that it is his determination to nationalise the hospitals—make them free—receive no payment from any patient, and admit the rich as well as the poor. This is not the first time that the Minister has said this, and it has not been repudiated by the Government or his colleagues. Indeed, it has been announced in bold headlines in the newspapers that the hospitals will be for the rich as well as the poor. I cannot imagine such a scheme being submitted by any responsible Minister. It is wanting in wisdom; it is devoid of statesmanlike grasp of the far-reaching consequences of such a proposal if carried out. It is repugnant to the principles of justice. (Hear, hear.) Right through the ages the hospitals have been the bright spots in the history of humanity. They have stood for all that is merciful, kindly, compassionate. They have been for the poor only, and it is because, Sir, they have been for the poor, that the association of the profession with them will redound to the profession's everlasting honour. Under the Minister's proposed scheme this happy relation will be abolished, the position of doctors and patients, the public, and the nurses, will be entirely altered. As sure as we are in this hall to-day, if the rich find admission to the hospitals, the poor will be elbowed out. As long as human nature is as it is, the influential will find a way of obtaining for their friends advantages and privileges denied to others not so fortunate, and always at the cost of the latter. Why, Sir, even now we occasionally have a man of political influence, or a woman of wealthy connection, admitted to the hospitals. And what happens? Claims are at once made on their behalf at the cost of the poorer patients. Sometimes even notices are sent round by "those who must be obeyed" (or think they must), that the routine and rules and regulations of the hospital are to temporarily lapse so as to give these people a special and unfair advantage. If this is so at the present time, when cases are isolated, what will it be when a large number of people belonging to the wealthy and influential classes have access to the hospitals? Metaphorically speaking, it can only come to this—that the poor will have to rest content with the crumbs of medical attention that fall from the beds of the rich.

So much for the want of wisdom of this proposal and its prejudicial effect upon the sick poor, for whom the hospitals have hitherto existed.

Let me now touch upon its far-reaching evil consequences to the community generally.

In one of Mathew Arnold's essays he compares the great poets of all nations to prophets or seers, because they have an intensity of insight, a discernment and clarity of vision denied to ordinary mortals. You may search through them all; but you will not find one who has not extolled the virtue of independence for the race and for the individual. The great Scottish poet and seer, Robert Burns, urges his countrymen,—

> To gather gear by every wile that's justified by honour.

Not for this or that purpose, but "for the glorious privilege of being independent." It is this glorious privilege that the Minister is endeavouring to teach the well-to-do people of Australia to throw aside as of no value. Has ever a falser lead been given by one in authority? And when we turn to the historians, the men who have given their lives to telling the story of the nations and the causes which led to their rise, progress, and decay, we find a unanimity, that no one cause for the decay and crumbling of a nation has been more potent than the loss of self-reliance and independence in the units composing it. Ignoring all this, ignoring the counsel of the wise, the teaching of history, the Minister does not hesitate to urge the well-to-do classes to go into the public hospitals and obtain medical attention at the cost of the State, that is, at the cost of the energetic, thrifty, and prudent members of the public.

Now let us consider this proposal in the light of the injustice it works to the medical profession. The resources, the unlimited resources of the State, are to be used to rob the doctor of his patient. The Minister says: "Oh, I will pay all doctors." I wonder what he means by that? There are only a small number of doctors, after all, who are connected with hospitals, and what will become of those outside whose patients are in this way taken away from them? Let me remind him that a needy, necessitous profession, with lowered ideals and status is a great danger to the public.

As to the value of the services rendered by the honorary medical staffs, the value of these gratuitous services, at the lowest estimate, amounted last year, in Sydney Hospital alone, to a quarter of a million sterling. Is the Minister going to pay the honorary medical staffs as at present constituted, or is he going to dismiss a large number of the members of the different honorary medical staffs and leave the regular work of the hospital to one-sixth, or one-fifth, or one-fourth of the present number? In that case the patients must seriously suffer. You cannot have three or four "know-alls" successfully carrying on the medical, surgical, and special work of a large hospital. There must be honorary specialists in every department or the hospital will be incomplete, and patients sent in for one disease and found to be suffering from another will not have the requisite first-class expert skill and knowledge to help them to recovery. If the staffs are to be as large as at present, and all to be paid, then the cost will be a burden to the State greater than it can bear.

The system of medical attendance in vogue in the chief hospitals of this country has been shown, by the experience of the civilised world, to be the very best possible. That is, after their University course, where they learn the theoretical part of their profession, the young graduates go as residents in a hospital, where they acquire their practical experience under the guidance of senior residents and the Honorary Medical Staff. After a year's practical experience, some leave and carry the knowledge gained to various parts; others remain for a longer term and join later in the Honorary Medical Staff as honorary assistants, serving thus a prolonged apprenticeship which fits them to succeed the senior Honorary Medical Staff, when, in the course of nature, these step aside.

If the working-classes, and those who represent the working classes in Parliament, support the Minister in this foolish and unjust proposal, they will throw away their most valuable possession, one that has been handed down to them for generations, and they get nothing in its place. But, whatever they do, and whatever the Minister does, this profession will do its duty—it will fight to the last ditch a proposal which is not alone intensely adverse to the interest of every medical man, but also intensely adverse to the interests of the sick poor, and, indeed, of the whole community, whose welfare is inseparably bound up with that of the medical profession. (Loud applause.)

I now have much pleasure in moving the following additions to the resolutions which were unanimously carried at last Congress:—

> "If it be found necessary by the various Governments to undertake the entire financial support of the hospitals, this Congress affirms its strong belief that it is in the interests of the sick poor, as well as the general community, patients whose circumstances enable them to obtain medical services outside the public hospitals should be excluded from these latter."
>
> "Unless such a provision is included in any Act to nationalise hospitals, the medical profession must reconsider its position with regard to its association with hospitals."
>
> "The Congress also is strongly of opinion that it is in the best interests of the hospitals and their patients that boards of management should be retained in the event of any alteration of the present system of financial support."

Dr. BARRETT: Mr. President, Ladies, and Gentlemen,—In seconding this resolution on behalf of the Committee, I desire to remind members that since the last Congress this movement to which Dr. Worrall has referred has advanced very greatly. The proposals of the Government are very complicated, and, if you will permit me, as briefly as possible I should like to explain them. In the first place the Government intends practically to do away with voluntary contributions to hospitals; the Government shall find the whole of the revenue, and all boards of advice shall be dispensed with. We are entitled, as citizens, to differ from that view; it would be better to keep up the present system of voluntary contribution, and we think it would be wise also to keep the Boards of Management. The value of these Boards of Management to a young country like this are almost incalculable, both in regard to the material services they render, and the sympathy they show with the unfortunate. At the same time, if Parliament, in its wisdom, decides that it will have neither Boards of Management nor voluntary contributions, they must perforce go, however strong the protests we may lodge. With regard to the proposal to admit all patients to hospitals, irrespective of their means, that is a point we must all most strongly object to. We must make proper and dignified and forceful representation of the unwisdom of such a policy. But I would point out, as was pointed out at the last Congress, that there devolves upon us the responsibility of providing intermediate accommodation for patients—for patients, that is, who are not destitute, but who are not well-to-do—and if we make such a provision on our own initiative, then our remonstrances are much more likely to carry weight. We say the hospital should be kept for those who have been unfortunate in life. That seems to be a sound proposition; but a constructive policy of providing for the intermediate portion of the population is equally necessary. With the report, I am in most complete sympathy; but I think it would be wise to hear what the Government has to propose, before taking any definite action. It would be wise to deal with it quite dispassionately, and to seek interviews with any Government which proposes such alterations, representing the matter from our point of view—a point of view, you may be certain, the Government would not understand. By being reasonable, temperate, perfectly fair, understanding the matter quite clearly, dealing with it in a clear light, we are likely to do more good to the community at large, and at the same time, maintain the self-respect and dignity of a great profession.

Dr. W. T. HAYWARD (South Australia): As a member of the Committee, coming from South Australia, I should like to say that the report read by Dr. Worrall meets with the approval of the medical practitioners of my State. Dr. Worrall and Dr. Barrett, in their extremely eloquent speeches, have put before you what might be called the higher politics—using the word "higher" in a double sense, perhaps—in connection with the conduct of hospitals. It is my opinion that the abuses at the present time are not likely to exist in the course of a year or two. Some time ago I had an opportunity of going into this question on behalf of the Adelaide Hospital (of the Board of Management, of which I am a member), and I came to the conclusion that the seat of part of the abuse was in the fact that payments were made by patients. I think that ought to be very definitely impressed on all members—the evil of the payment for services. Another point that came out very strongly in my investigations was that the greatest sinners in regard to sending unsuitable patients into hospitals were the members of the medical profession. When we had looked into several definite cases that had been brought before the Board of Management in regard to hospital abuses, we found that these cases had been recommended by medical men; so I ask the members of the medical profession here assembled to be extremely careful in sending these recommendations. It is a most awkward thing, when you get up on a Board of Management and protest against such and such patients being admitted because they are not proper subjects, to find that the order has been sent in by a member of your own profession. The trouble, again, is that patients who came into the hospital and pay these fees, have unfortunately the idea that they are paying for services that they receive. I have a recent example in this connection which occurred only a week or so ago in my own State. A relative of a very prominent member of the Government——

MEMBERS OF CONGRESS: Tell us who it was.

Dr. W. T. HAYWARD: It was the daughter of the Premier of South Australia. She was sent down to the Adelaide Hospital to be operated on for appendicitis. (Cries of "Shame.") That recommendation was made on the advice of one of the highest medical officials in the State, and the order was signed by a member of the staff. You see, therefore, that, as a profession, we have to bear the onus of this sort of abuse to a certain extent. I am perfectly certain—I do not know the Premier personally—that he is not such a fool as to put himself in the position that he is receiving charity. He does not think for a minute he is receiving charity, though he is certainly doing so; and while the present state of affairs exists, not only have we the Premier, but every other member of that class taking advantage of the hospital. Such a thing, is decidedly an abuse. I hope, gentlemen, we shall stick very strongly to these resolutions, and do our best to prevent such a thing happening in the future. (Applause.)

Dr. NASH: Mr. President, Ladies, and Gentlemen,—At the last session of Congress in Melbourne, I stated that the basic principle of the treatment of patients in public hospitals should be that the people who went there were unable to pay; (hear, hear) and that no fees of any kind should be charged from patients admitted within their walls. Now, those of you who were at that session may remember that a patient was admitted to the Sydney Hospital who paid £3 3s. a week, and I promised the session of Congress that I would make inquiries, because I, as a member of the board, knew nothing of it. Now, I have kept my promise, and at the first available opportunity, I made inquiries as to the patient, as to the payment, and as to the doctor who sent the patient there. Now, I might tell you that I have never been able to speak about it at the Board since, because the Secretary of the Board told me that the patient was Dr. Worrall's; not only that, but Dr. Worrall sent the patient in, and the patient was well known to him. Well, ladies and gentlemen, what could I do?—because it was Dr. Worrall who made the very statement against the practice at the session of Congress. I could do nothing with the Board of the Sydney Hospital; but does it not fit in with what Dr. Hayward has just said—that the greatest sinners in this regard are the members of the staff? That is a personal explanation, because I promised the Congress that I would give it, and I have given it. (Applause.) It is no good the medical profession making protests that things are to be so-and-so, unless they are prepared to back them up by their own actions. (Hear, hear.) And they must back them up by their own actions when it may be that apparently their own personal interests are going to suffer. We cannot bring anything about that will be satisfactory to our profession while personal interests are allowed to step in and lead us to do actions which we know, based upon sound principles, are absolutely wrong. Now, ladies and gentlemen, I ask you not to be influenced too much by what my friend, Dr. Worrall, said about the suggested alterations in the conduct of hospitals which have been appearing in the newspapers, because our present Minister is a man who wishes to do what is right. He wishes to do what is right by the public; he wishes to do what is right by the profession; and there is nobody who knows him, in this State or anywhere else, as a public man, better than I do. I say it will be wise, both in the interests of the profession and of the public, to wait until we have an opportunity of getting some absolutely definite statement from that Minister as to what he wants to do.

A MEMBER OF CONGRESS: He has said it twice.

ANOTHER MEMBER: Three times.

Dr. NASH: You have seen it reported badly in the newspapers; that is all any man has seen, because he has never yet said it in a place where he was responsible for what he said. (Cries of "Oh!") Let me finish. Let me say this: He has never said it in a place where he was face to face with any man who could bring him up to the scratch to what he said.

A MEMBER OF CONGRESS: He has said it, anyhow.

Dr. NASH: Never mind. We will get him some day. I shall be in a position, when he says the thing authoritatively, to ask him what he means, and I promise you, gentlemen, that when the opportunity occurs, I shall not be backward in asking, in the interests of the profession, what he does mean. (Hear, hear.) Rest assured, I will do it all right. If I get the opportunity, I will do it.

A MEMBER OF CONGRESS: Let us know it at next Congress.

Dr. NASH: You will find, if it is reported correctly, matters will not work out exactly as he thinks.

Now, with regard to the question of contributions, a peculiar position has arisen in the State of New South Wales, in this respect, that the Government finds nearly all the money for the hospitals.

A MEMBER OF CONGRESS: Half.

Dr. NASH: A good deal more than half. Out of the £25,000 or £30,000 that the Sydney Hospital and the Prince Alfred Hospital will spend during this year, over £20,000 will come from the Government; and, mind you, that is only for maintenance and upkeep. The Government of this State provides money for building and equipment and various matters like that. (Cries of "No, no!" "nonsense!") I tell you it is so. What I say with regard to the Prince Alfred Hospital and the Sydney Hospital, which are the great hospitals of the City, is an absolute fact. I know it. They find practically all the money for building purposes, and they provide over three-fourths of the money for maintenance. I am speaking from what I know, because I happen to be a member of the Board of the Sydney Hospital. That puts the hospitals in a very peculiar position when you begin to say nasty things about the Government, because you are attacking the very concern that is keeping the institutions going. I do not want to continue long. I just want to tell the members of the Congress the facts of the case in regard to this matter. I should like the Congress, and all meetings of medical men, to stand up to what I believe to be the first principles which should guide the profession in their dealings with public hospitals, and that is what I started with. They should insist that nobody should be admitted to a hospital who can afford to pay. Nobody going into a hospital should be allowed to pay. (Hear, hear.) That is the fundamental principle of all my arguments and my talk. I think it is the standpoint from which the medical profession should always think the subject out. They should insist upon it. I have found in my battles in life, and they have been a few, that so long as I was in possession of the hard basic facts, which were right, and which have been proved time and time again to be right, that I could always come back to it, no matter what superficial arguments and thoughtless suggestions and statements were made. I always come back and say: "Well, the principle is that there must be no payment," and we, as a profession, should say this, that the moment there is payment, then, the highest work, the best work, the most exalted position of the medical profession is taken away from beneath its feet, because the best work is done for these poor people—the most satisfactory work, anyway; work which is of the most benefit to the whole of the community and the profession, work which must always be done in the hospitals, where the poor are treated, and where nobody is charged. (Applause.)

PRESIDENT: Dr. Worrall wishes to make a personal explanation.

Dr. WORRALL: I want to say at once that there is not a shadow of foundation for the statement that I sent that patient into the hospital. The patient was in my bed. I mentioned that the patient had been sent in under me. She was admitted by the Superintendent. I discovered who she was while she was in the hospital, and I told her if anyone else but herself had informed me, I would not have believed it, and I did not say "Good morning" to her afterwards. (Applause.)

Professor ALLEN: The argument has been placed before us that because the Governments of Australia pay a large part of the cost of building hospitals and a large part of the cost of their upkeep, the Governments should, therefore, take over the hospitals altogether and administer them as State institutions without independent Boards of Management, without any voluntary contributions whatsoever. That seems to me an entirely false argument. If you will only think it over from a few other standpoints, you will not fail to see what it means. We are assembled to-day in the Great Hall of a University. How are universities maintained? What have been the great sources of the revenues and the building funds of the University of Sydney? Do we not remember the Challis Bequest, the Fisher Bequest, and all the other benefactions which have done so much to bring this great institution to its present state of prosperity and efficiency. Take, as another example, the Melbourne Hospital. Who provided the funds for the complete rebuilding of the hospital which is now in progress? Not one penny has come from the public funds. The whole expense is borne by private benefactors. If the Governments interfere in any way that has been proposed, and nationalise the hospitals, the action will not end there; it will spread to the universities, and to countless other institutions, and the whole stream of private benevolence will be dried up. (Hear, hear.) Then I would ask: What does this nationalisation imply in other respects? How does it affect the general status of the medical profession, its ideals, its aims, its independence? Think over that. Consider what it will mean to us, as members of the great corporate body of medical practitioners who are striving, according to their abilities, to lead lives of independent effort and noble aspiration. Again I would ask: What will be the reflex influence of nationalisation on the medical student of the future? Will it tend to encourage him to prepare for a life of lofty professional work, for a life full of self-sacrifice, or will it lead him to cultivate the arts of the log-roller? And again, what will be the result on medical research, on scientific progress? What will be the outcome to the many great mutual provident organisations that have done so much to provide medical aid for the thrifty members of the working classes in the community? Think of the enormous issues involved. In the United Kingdom, at the present time, how anxiously the friendly societies are watching their position and considering what the future will bring. I ask you to think what will be the influence of this proposal on all forms of charity. The very basis of charity is affected. Will the change be for good, or will it be for evil? These are some of the great questions that should be carefully thought to a conclusion. We have not time now to work out any of them in detail. They are not matters that can be treated fully in a meeting such as this. Here we can only lay hold of some great principles, and make up our minds stoutly to adhere to them. (Loud applause.)

Dr. FRESHNEY: I wish to say a few words on this. For the past seventeen years I have been connected with the Toowoomba Hospital. During that time it has gone on pretty well. I have some figures here which might be interesting. In 1901 we had 1,078 patients per year; last year 1,091. In the intervening years we used to run up between 1,200 and 1,400 patients. I induced the Committee to put in a resident secretary, the result of which was that the number of patients was reduced almost to what it was ten years ago. In the two years before that appointment was made, £826 was paid by patients; in the two years after, £1,464 was paid by patients, showing the necessity of looking into the financial position of those seeking admission. Well, now, up there, we have this system:—A resident paid surgeon, with myself as superintendent, and an honorary staff. We have there the right of charging patients who pay the full fee, 28s. per week; and then we can charge a fee in accordance with their position in life. That, I think, is not a bad scheme, because, in country districts, you cannot lay down hard and fast rules in regard to keeping out patients who can pay a little. It is much better to look into the financial position of the patient and get the medical men paid. That is all I want to say.

Dr. JAMES HOPE (Western Australia): I come to speak from the far country, Western Australia, where you cannot deal with things as you can here. We have to deal with people in various positions of life. We have there people who are well able to afford and who are supposed to go into private hospitals. We have people who can afford to pay a hospital charge but who cannot afford to pay the ordinary medical fees, and we have the poor people who can pay nothing. You cannot lay down hard and fast rules for people, not paupers, who are able to pay hospital fees. I know there is a great deal of imposition from time to time; but medical men in country districts are obliged to bring patients in who are living at considerable distances. They are not able to pay fees in the ordinary sense, and, therefore, the doctor must be broad-minded and allow a certain class of people to come in and support the hospitals. To disallow such a privilege to those people living in Western Australia, a very sparsely populated country, would inflict a hardship. It is necessary to provide them with comfortable hospital accommodation.

In the gold-fields, I have heard, in the report of the Committee on Hospital Management, that the men pay there 1s. a fortnight; but it is 1s. a week in the gold-fields hospitals. With the exception of these places, most of the hospitals are managed by Government medical officers. If a person is able to pay, and the conveniences of his house are such that he cannot have the proper nursing at home, he is admitted to a hospital, where he pays the hospital fees and the fee of the doctor, too.

A MEMBER OF CONGRESS: Does his own medical officer attend him in that case?

Dr. J. LOCKHART GIBSON (Queensland): As a member of the Hospital Committee, I should like to say a few words in support of the resolution submitted by Dr. Worrall and Dr. Barrett. There are three things which should be remembered. The first is that we must impress upon the public and statesmen alike, with all the earnestness in our power, that the results we are trying to arrive at at this Congress are for the general good of the community. There is no use trying to pass any legislation that is not for the benefit of the public in the first instance, and I am firmly of opinion myself that these resolutions are for the benefit of the public who take advantage of the hospitals, and for the public who are paying for medical assistance outside hospitals. The second point I wish to make is that no patients' payments be taken in any public hospitals in large towns. The third is that any legislation which is passed, whether the hospitals are to be continued to be managed as they are managed at present, and supported by public subscriptions, or whether they are taken over by the State, no patient be admitted to any public hospital in a large town, at any rate, who is in a position to pay for medical services outside. That is a detail that can be managed quite properly by making country hospitals in certain districts, a combination of public and private hospitals. That does not involve any alteration in these resolutions.

With regard to Dr. Barrett's suggestion, the suggestion put forward at the last Congress in Melbourne, as to intermediate hospitals, that also is a detail, and whether they are established by the profession or by the public is a detail also. Intermediate hospitals are certainly necessary. At present the rich are getting the best attention by paying for it; and the poor are treated as well at our public hospitals: intermediate people are less well off than either.

I should like to endorse most cordially the remarks of Professor Allen. We are all agreed that, if possible, the hospitals should not become Government institutions, and these resolutions, providing for what should be done if they do become Government institutions, are not meant to be taken as approving of their nationalisation. I was glad to hear Professor Allen speak as he did, because it was just the speech we wanted, and, I think, coming from him, it will do an infinity of good.

Dr. E. ROBERTON (New Zealand): Coming as I do from New Zealand, a land of experimental legislation, a land responsible, to a great extent, for the trend of modern legislation so far as regards hospitals, I want to emphasise mainly one thing: that when legislation towards the nationalisation of hospitals is taken in hand by governments, the very first thing recognised is that the hospitals are established for the sick poor, and there is always a special provision providing definitely for that. The debates on the Act under which we are at present working, the Act of 1885, showed most definitely that the State hospitals were intended for the sick poor. Unfortunately that was not definitely stated in the preamble to the Bill, but it was taken for granted later on. As our country became more democratic, demands were made by the rich for the establishment of hospitals, and other demands were made by friendly societies. It became a political question practically every three years, for the capture of votes. A certain class of politicians aims at finding out what people want and giving it to them, because they know by that means they can be sure of getting votes, and votes are everything to them. A good deal of political jobbery has been going on in New Zealand in this connection; but the outcome of it all has been that the hospitals are universally recognised as existing for the sick poor, and they are now in such a position that I doubt if it would be possible, without a very great struggle, to effect any change in that direction.

Dr. Crowther: I will not keep you a moment. One thing you must not forget; if you want to do anything you must organise, organise, organise, and demand your own terms.

Dr. Worrall: I am very grateful at the way my remarks have been received.

President: I will now put the resolution moved by Dr. Worrall, and seconded by Dr. Barrett.

Carried unanimously.

REPORT OF THE COMMITTEE APPOINTED TO REPORT ON SYPHILIS.

Dr. Corlette: Mr. President, Ladies, and Gentlemen,—I beg to move the adoption of the report of the Committee appointed to report on Syphilis, and I hope you will allow it to be taken as read. It is, perhaps, unnecessary to say, in submitting this report, that we have had a very difficult subject to deal with, a subject bristling all over with controversial points. I do not think any member of this Committee expected that the report, when completed, would satisfy everybody. The report might have been made more controversial, or it might have been made less controversial. Anyhow, we did the best we could. I think we are all agreed on what is in the report, if we are not all agreed as to what has been left out. That is all I think I need say about this. I hope you will not be too dissatisfied with the smallness of the material which you find in this report. It is not a subject which can be completed in one year or two years; it is a subject which would have to be increased during many years before we could get on to the stage to which we would like. I beg, therefore, to move that this report be adopted.

Professor Allen: As one who was concerned with this subject at the last session of Congress, I should like to say I appreciate very much the practical wisdom the Committee has shown in the form that it has given to this report to-day. I think that we can all adopt what it says without question or doubt. As to anything further, the matter can be carried on. When we have a debate to-morrow in connection with the section of public health, when that subject and many other subjects resulting from the recent investigations that have taken place in Melbourne will be brought before the members of Congress, I shall draw the attention of those who may not be familiar with the facts to the very important reports that will be submitted, dealing with 5,000 cases, which were very carefully investigated and reported upon in Melbourne during one twelve months of work. Dr. Ham, of Melbourne, will submit the remedial and preventive measures to the Congress at the meeting.

The motion was seconded, and carried unanimously.

REPORT OF COMMITTEE ON SYPHILIS.

This Committee met for the first time on Friday, June 10, 1910, and on many subsequent dates up to the present, and have considered the subject of Syphilis under the two heads—

1. Prevalence of the disease.
2. Measures which might be adopted to diminish such prevalence.

1. PREVALENCE OF THE DISEASE.

Much consideration was given to the question of how best to obtain reliable data on this point. One method suggested was to examine the Wassermann reaction of a large number of hospital patients at the Metropolitan General Hospitals, in order to ascertain what proportion gave a positive result. But it was thought by the Committee that there were so many possible sources of error in such an investigation that no deductions of value could be drawn from the results obtained thereby, and accordingly this plan was not adopted.

By the courtesy of the Inspector-General of the Insane, however, the Committee has been furnished with the results obtained at the Hospital for Insane, Callan Park, where the Wassermann test has been applied to the blood of all cases admitted since 1st June, 1910. This work has been under the supervision of Dr. J. F. Flashman. Following are the results:—

Admissions—1st June, 1910, to 31st July, 1911	475
Bloods tested for Wassermann reaction	452
,, Positive result obtained in	65
Percentage	14·4

More promising appeared the proposal to address the medical practitioners of the metropolis, asking them to keep a record for the six months, 1st October, 1910, to 31st March, 1911, of all cases of Syphilis seen by them during that period. Accordingly on 26th September, 1910, a circular to this effect was issued to all medical practitioners within the metropolitan area, and with the circular a form on which such cases were to be tabulated as Primary, Secondary, Tertiary, and Congenital Syphilis, and distributed under Age period, Sex and Social State divisions. At the end of the six months a second circular was issued, asking for these returns.

The response to these circulars was disappointingly meagre. Only twenty returns were received from private practitioners, which may be summarised as follows:—

	Age.								Social State.						Totals		
	5 yr.		5–15.		-3 .		Over 30.		M.		S.		W.				
	M.	F.	M.	F.	M.	F.	M	F.	M.	F.	M.	F.	M.	F	M.	F.	M. & F.
Primary Syphilis	...	...	...	...	36	3	10	...	11	...	33	1	2	...	46	3	49
Secondary ,, ...	1	...	...	...	61	28	29	2	14	18	39	7	2	...	91	30	121
Tertiary ,, ...	...	...	1	...	10	2	49	29	24	22	20	1	2	5	60	31	91
Congenital ,, ...	9	12	1	3	3	2	...	...	...	...	...	...	...	...	13	17	30
All Forms ...	10	12	2	3	110	35	88	31	...	...	...	...	...	...	210	81	291

In addition, the following hospital returns were received, some of which, it will be noticed, are for the six months, 1 January—30 June, 1911:—

Royal Prince Alfred Hospital—In-patients and Out-patients, 1 October, 1910—31 March, 1911.

	Age.								Social State.						Totals.		
	5 yr.		5–15.		15–30.		Over 30.		M.		S.		W.				
	M.	F.	M.	F.	M.	F	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M. & F.
Primary Syphilis	...	...	...	...	2	4	1	...	...	...	3	4	...	...	3	4	7
Secondary ,, ...	...	...	...	...	15	56	12	28	8	43	7	35	2	6	27	84	111
Tertiary ,, ...	...	...	1	...	9	11	16	18	9	12	16	11	1	6	26	29	55
Congenital ,, ...	6	4	...	1	1	4	...	1	...	...	7	10	...	...	7	10	17
All Forms ...	6	4	1	1	27	75	29	47	17	55	33	60	3	12	63	127	190

The Sydney Hospital—Out-patients, 1 January—30 June, 1911.

Primary Syphilis	...	...	1	...	47	8	42	7	...	12	...	2	...	1	90	15	105
Secondary ,, ...	...	...	...	1	13	3	15	7	...	8	...	2	...	1	28	11	39
Tertiary ,, ...	...	...	1	...	11	6	33	15	...	17	...	1	...	3	45	21	66
Congenital ,, ...	4	...	1	...	1	...	...	1	...	1	6	...	...	...	6	1	7
All Forms ...	4	...	3	1	72	17	90	30	...	...	...	...	...	...	169	48	217

THE COAST HOSPITAL—In-patients, 1 October, 1910—31 March, 1911.

	Age.								Social State.						Totals.		
	5 yr.		5–15.		15–30.		Over 30.		M.		S.		W.				
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F	M.	F.	M. & F
Primary Syphilis	...	...	...	...	3	...	1	...	...	...	...	...	...	...	4	...	4
Secondary ,, ...	...	...	...	...	53	2	28	2	...	...	...	...	...	...	81	4	85
Tertiary ,, ...	...	...	...	...	8	1	13	...	...	...	...	...	...	...	21	1	22
Congenital ,, ...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
All Forms ...	...	...	...	...	64	3	42	2	...	...	...	...	...	...	106	5	111

ROYAL ALEXANDRA HOSPITAL FOR CHILDREN.

Dr. Litchfield's O.P. Dept., 1 January—30 June, 1911.

Congenital Syphilis ...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	35

ROYAL HOSPITAL FOR WOMEN, PADDINGTON.

1 October, 1910—31 March, 1911.

Primary Syphilis	...	...	...	...	...	2	...	...	...	...	...	...	...	...	...	2	2
Congenital ,, ...	3	5	...	...	...	...	...	...	...	...	...	...	...	...	3	5	8
All Forms ...	3	5	...	...	...	2	...	...	...	...	...	...	...	...	3	7	10

NOTE 1.—In the Sydney Hospital statistics, the Committee are informed that cases recorded mere as "Syphilis" were included as "Primary Syphilis."

NOTE 2.—The Coast Hospital has a special ward for in-patient treatment of males suffering from Syphilis, and the Royal Prince Alfred Hospital has similar accommodation for Syphilitic females. The Sydney Hospital excludes cases of Syphilis from the wards; those needing indoor treatment are therefore drafted into either the Coast Hospital or the Royal Prince Alfred Hospital.

It is obvious that these data are not sufficiently ample to justify any exact conclusions as to the incidence of Syphilis on the population of the metropolis, but at least they confirm the belief that it is a disease of considerable prevalence.

That the prevalence of Syphilis and other venereal diseases is, however, diminishing, at least amongst the men in the Navy, is indicated by the memorandum of Fleet Surgeon Vidal G. Thorpe, R.N. (a member of this Committee), which is attached. From this it will be seen that the incidence of Primary Syphilis among men of the Royal Navy serving on the Australian Station has fallen from 28 per 1,000 in 1905 to 6 per 1,000 in 1910. Of the total cases of venereal disease that occurred in 1910, 66 per cent. were infected in Sydney, but it should be mentioned in justice to this port that this "bad pre-eminence" is due to the fact that Sydney is the headquarters of the Station, and that in consequence more time was spent there than in other ports.

2. MEASURES THAT MIGHT BE ADOPTED TO DIMINISH THE PREVALENCE OF SYPHILIS.

This subject was very carefully considered by the Committee. The principal measures that might be adopted fall under the following heads:—

1. State regulation of prostitution.—This, as exemplified by the Contagious Diseases Acts, did not commend itself to the Committee. The Committee desire, however, to record their hearty approval of the Prisoners' Detention Act of 1908 (N.S.W.), which came into force on 1st January, 1909, and under which certain prisoners whom the Gaol Surgeon certifies to be suffering from venereal disease, may be detained in a "lock hospital" until free of such disease; and of the Police Offences (Amendment) Act, 1908 (N.S.W.), which *inter alia* imposes penalties on "common prostitutes" who solicit or importune for immoral purposes any person who is in any public street, thoroughfare, or place.

The Committee believe that these two measures are calculated to be of great value in diminishing venereal disease.

2. Notification.—The Committee were unable to agree on a recommendation of compulsory notification of Syphilis. It was, of course, admitted by all that the information so gained would be of the utmost value, but the view was expressed that the fear of publicity consequent on such notification would deter patients from consulting their ordinary medical attendant, and would cause them either to neglect treatment, or to seek it from ignorant and irresponsible quacks.

3. Increase of Facilities for the Treatment of the Disease.—The Committee were in hearty accord as to the desirability and value of this measure, and wish to express their concurrence with the views put forward by the Hon. Dr. Mackellar, M.L.C., in a speech on this subject in the Legislative Council of New South Wales, 6th July, 1911, in which he urges that the disease should be treated not in special Lock Hospitals, admission to which must necessarily involve a stigma on the patient, which may deter him or her from resorting to such hospital, but in special wards or departments of one or more of the general hospitals.

In view of the now proved efficacy of the new remedy Salvarsan in rapidly healing those surface lesions which are the most fertile sources of contagion, it seems not too much to hope that the systematic application of this treatment would have a very potent influence in reducing the prevalence of the disease. If this be so, it would be in the best interests of the State to make this treatment available to all syphilitics.

In this connection reference should be made to the proved efficacy of Calomel ointment as a prophylactic against syphilitic infection.

4. Prohibition of Entrance into the State of Syphilitics in an active stage of the Disease.—This is already provided for under Commonwealth legislation, and it appears unnecessary to say more here than that the Committee heartily concur in such prohibition, and recommends that it be rigidly enforced.

Less direct in their application, but, in the opinion of the Committee, of great importance as factors in diminishing venereal disease, are Temperance and Early Marriage. It is recognised by all who have to deal with them that a large proportion of venereal cases incur the risk of infection through being intoxicated, and consequently for the time being deprived of their normal self-control. The statistics of the British Army show a decline in venereal disease coincident with a spread of temperance, and we may confidently expect the same in the civil community also.

The value of early marriage as a prophylactic measure is self evident, though it is not perhaps within the scope of this Committee to suggest measures to deal with the economic problems involved in this question.

In concluding this report, the Committee might perhaps be permitted to express the opinion that Gonorrhœa, as a factor in damaging the health of the community, is of equal, if not greater, importance than Syphilis, and to suggest that this disease might well be the subject of consideration at this or some future Congress.

MEMORANDUM ON VENEREAL DISEASES IN THE AUSTRALIAN SQUADRON.

By Fleet-Surgeon Vidal G. Thorpe, R.N., M.R.C.S., Eng.

It will be seen from the following table that venereal diseases amongst the men of the Royal Navy serving on the Australian Station have steadily decreased in numbers since the year 1906, when the case ratio for all forms was 167 per 1,000:—

	Ratio per 1,000.			Totals.
	Chancroid.	Syphilis—primary.	Gonorrhœa.	
1904 ...	...	22	101	123 per 1,000
1905 ...	10	28	91	129 ,,
1906 ...	19	18	130	167 ,,
1907 ...	24	24	102	150 ,,
1908 ...	20	13	87	120 ,,
1909 ...	8	7	68	83 ,,
1910 ...	6	6	63	75 ,,

In the year 1910, Sydney was responsible for 66 per cent. of the cases, Melbourne and Hobart 11 per cent. each, Auckland 4 per cent., and the remaining 8 per cent. of cases were distributed between seven other cities of Australia and New Zealand.

I think that we must acknowledge that we are greatly indebted to the Government of New South Wales for passing the Prisoners' Detention Act of 1908, and that it undoubtedly has been a potent factor in the diminution of venereal disease amongst the men of the Australian Squadron. The fall in numbers was marked during the two years following the passage of the Act, especially as regards Chancroid and Syphilis, the case ratio per 1,000 falling from 20 to 6 in the former disease, and from 13 to 6 in the latter.

UNIVERSAL (MILITARY) TRAINING.

Dr. David Thomas: At the request of the Council of the British Medical Association, I have pleasure in moving the following resolution:—"That the minimum fee payable for the examination of Cadets, under the Universal (Military) Training System, should be 2s. 6d. for each such examination." Since I have given notice of this motion, there have been certain regulations issued by the Minister of Defence, which may or may not meet with the views of the profession. Dr. Fetherston, from Melbourne, has notified me that the Victorian Premier seems to be inclined to adopt these regulations, and, with the view of facilitating the business of this meeting, he has certain proposals to make with a view of reporting to a meeting on Saturday next, which, I think, would meet with the views of the Congress, and probably expedite matters considerably. Whether it is the wish of the meeting to discuss this important question now, or whether it will leave it to be reported upon until next Saturday, when Dr. Fetherston proposes that a certain Committee report upon the new regulations issued by the Minister, is for the meeting to say. I simply, at the present moment, formally move this resolution.

Dr. Davenport seconded.

Dr. Fetherston: It seems very simple that 2s. 6d. for the examination of each recruit should be paid; but it is not quite so simple as it seems. I would suggest that a sub-committee or a committee be formed to go into the new scheme just recently proposed, and at the same time adopt Dr. Thomas's suggestion, and bring up the report next Saturday. I think a good deal can be done to help the Area Medical Officers by this Congress, if we bring it up to some shape whereby we can go to the Minister or to the powers that be, and let them know exactly how we view the matter. I propose that the committee shall consist of Surgeon-General Williams, Col. Roth, Col. Eames, Dr. David Thomas, Col. Ramsay Smith, Col. Sutton, Col. Giblin, Dr. P. Seed, Lieut.-Col. Fetherston.

Dr. Stoker: I second that.

President: I have a letter from the Secretary of the Australian Club, which reads:—"I am directed by the President and Members of the Australian Club to request that gentlemen members of your Congress who are visiting Sydney from other States or New Zealand will do them the honour of considering themselves honorary members of the club until the rising of the Conference." I have also received a letter from the President of the Australian Jockey Club, offering to give honorary tickets to members and their wives from other States or New Zealand, who may be staying after the Congress, for their Spring Meeting, which takes place at the end of next week. Names must be sent in before the end of this week, if possible, to the honorary secretary of the Congress.

In regard to this afternoon's Government picnic, I have just received a wire:—"Please inform Congress each one attending excursion to-day will require obtain ticket from you. Cannot admit without tickets." In regard to the Caves trip, it is overfull. There has been a little mistake. The Government undertook to take members up to the number of sixty, including their wives or one lady who may be here from other States or

New Zealand. Some members have put down three or four ladies' names. The Government will not accept that. They wish as many members of Congress as possible to visit the Caves, but no member can take more than one lady. (A voice: "Quite enough.") The means of getting there are limited. A long distance has to be travelled by motor-car. As regards the picnic this afternoon, I should like to say that top-hats and frock-coats are not necessary. (Hear, hear.)

The Honorary General Secretary wishes me to announce that he is issuing tickets for the various dinners on Wednesday night, and any member who does not receive one by post to-morrow, is requested to communicate with him at once, when tickets will be sent to him. I should like the members of the Executive Committee to remain behind, as we have a very important matter to discuss, which will not take more than two or three minutes. I am mentioning this now, because I see a number of gentlemen are leaving.

A MEMBER OF CONGRESS: I should like to point out that Surgeon-General Williams has notified the Army Medical Corps that the sum of £60 per annum has been granted by the Minister of Defence for the examination of the cadets, and that this arrangement is only for the present financial year, and if, at the end of the financial year, it is found to be unsatisfactory, that further consideration will be given, and an increase possibly will be allowed. So that, at the present time, there is no necessity to refer this to a sub-committee. The General emphasises the fact that every consideration will be given by the Minister of Defence to the requests of this Board, and I do not think, under those circumstances, it would be in any way advisable to deal with it in any further way. I would suggest, therefore, that the matter be adjourned until we hear from Surgeon-General Williams.

ANOTHER MEMBER OF CONGRESS: Surgeon-General Williams stated on Saturday that he would be here.

DR. DAVID THOMAS: If Surgeon-General Williams is on this committee surely we can hear what he has to say.

PRESIDENT: Does the meeting agree to the withdrawal of the original motion? The amendment then becomes a substantive motion. It is that a committee, consisting of Surgeon-General Williams, Col. Roth, Col. Eames, Dr. David Thomas, Col. Ramsay Smith, Col. Sutton, Col. Giblin, Dr. P. Seed, Lieut.-Col. Fetherston, be formed to consider this matter and report to the meeting on Saturday morning.

Carried.

STATISTICS.

DR. FREDERICK WATSON: I beg to move the resolution standing in my name:—"(1) That it is advisable that all available Australian medical data and statistics from the year 1788 be collected and summarised. (2) That for this purpose a committee for each State be appointed. (3) That a central committee at Sydney be established to receive the reports of the State committees, and to arrange for the publication of all matter they think of value in such way as from time to time is desirable, and to report progress at next meeting of Congress." Further, I should like to move that the resolution standing in my name be referred to the section of Public Health, to report upon it at next Saturday morning's meeting.

The motion was carried.

POSTAL AND TELEGRAPHIC FACILITIES.

Dr. R. H. Todd, in the absence of Dr. Clarence Read, moved: "That inasmuch as in the opinion of this Congress every facility should exist to enable the sick and the injured to obtain medical assistance at any time they may need it—(*a*) means of communication by telephone and telegraph should be available by night as well as by day, even if a special charge is made for their use at night; (*b*) public letter-boxes, especially in populous places, should be cleared up to a late hour each night, or early enough each morning to allow of an early morning delivery to be made." This is a matter which really does not require very much comment. I should merely like to state the facts. In the past, as regards telephone and telegraph, there have been means of communication existing practically all night, when necessary, and also as regards the letter-boxes about cities and towns, they have been emptied at a late hour so as to allow of a delivery by first post on the following morning. The recent innovations made by the Postmaster-General have entirely changed all this, and letter-boxes, instead of being cleared at midnight, are cleared at about 9 or 10 o'clock; sometimes 9 o'clock in some places, 10 o'clock in others. Throughout New South Wales this has been found a great detriment to the sick poor and sick people generally. Doctors have not been able to get into communication with patients, nor have patients been able to get into communication with doctors. It is a matter that has been raised by some of the medical men in New South Wales. It was first raised by some of the members in country districts (which we know as the Western Medical Association) who moved a resolution much to the same effect as this one. It was felt that this would be a fitting opportunity to approach the Postmaster-General on the matter, and to urge upon him, in the public interest, the advisability, in the interests of sick people, of better postal and telegraphic facilities.

In regard to this matter, when coming to the Congress this morning, I read in the newspaper that provision has already been made by the Postal Department for sending urgent telegrams and urgent telephone messages after hours, in special cases, for an additional charge. Of course I do not know whether the Postmaster-General saw this matter was on the business-paper of our Congress. Anyhow, it appears that his attention has been drawn to the matter. We do not know what has been done definitely, so, therefore, I do not propose to alter this motion in any way, notwithstanding this report in the newspaper. I have much pleasure, Mr. President, ladies, and gentlemen, in moving that resolution standing in the name of Dr. Clarence Read.

The motion was seconded, and carried unanimously.

COMMONWEALTH NAVAL MEDICAL SERVICE.

Dr. B. J. Newmarch: I beg to move the resolution standing in my name. It is simply an opportunity given to the Congress to discuss the question of the forms of the Commonwealth Naval Medical Service. Admiral Henderson's report on the matter will come before them, and it will be an opportunity for the profession to offer suggestions to the Admiralty with regard to a Commonwealth Naval Board, so that once and for all we will have what I hope will be a popular service, and a service in every way agreeable to the profession.

> "That this Congress expresses its appreciation of the suggestion of the Officer in Charge of the Medical Branch of the Commonwealth Navy to invite discussion concerning the initiation of the Naval Medical Service for the Commonwealth of Australia.
>
> "That the letter conveyed by that Officer, together with the suggestions appended, be read by the Secretary of the Naval and Military Section before the Section, and a discussion invited.
>
> "That the result of the discussion be reported to Congress, and a full report and decision forwarded to the Officer in Charge."

Carried.

STANDING COMMITTEE—"COMMISSION OF CONGRESS."

Dr. J. Adam Dick: Arising out of two paragraphs in the President's report, I beg to move that a Standing Committee of this Congress be appointed, as detailed in the resolution standing in my name. That that is necessary, you will understand on brief consideration. During the next three years, probably, there will be introduced into the various States, or possibly into the Federal Parliament or the Dominion, measures that will affect the medical profession, and possibly a national insurance scheme, similar to that which has been introduced in the British Parliament, will be introduced here. It is advisable, therefore, that there should be some permanent body established, to which should be given powers to accept any scheme which may be introduced into the State or Commonwealth or Dominion Parliament. The suggestions seem to be long when looked at at first glance, but if you will go into them you will see they are drawn out very carefully, and in detail, by a member who understands legal forms. It is necessary to have them drawn up in this way, so that the Congress need not waste time discussing what powers and duties should be given to such a commission. The main thing is its necessity, and of that I am sure everyone is convinced. If no special body like this is established for the next three years, a scheme might be put forward to the Commonwealth, the Dominion, and the various State Parliaments, and we would have no large executive body to express our opinions and to see that the great interests of the medical profession were properly safeguarded. You all know it is through organisation, and organisation alone, that the home medical practitioners have been able to get their claims recognised. Had they not organised they would not even have been listened to by the Chancellor of the Exchequer. It behoves us, therefore, to take this opportunity—an opportunity we will not have for another three years, to establish a body that will have powers and duties given to it by this Congress, to look after our best interests, either in the Commonwealth, the Dominion, or any State. I, therefore, beg to move the resolution standing in my name,—

> That a Standing Committee of the Australasian Medical Congress, to be called "The Commission of Congress," be and is hereby formed with the following constitution, viz. :—

1. *Membership.*—Such Commission shall consist of the President; all Past Presidents, who shall signify in writing their consent to be members; all Past Honorary General Secretaries, who shall signify in writing their consent to be members, provided that, if there shall have been two or more Honorary General Secretaries in any one Session, and two or more thereof so signify their consent, the President, either by drawing lots or otherwise, shall determine which one shall be a member, and the other or others shall thereupon be excluded; three members appointed by the Council of each Branch in Australasia of the British Medical Association, provided that the President shall cause each such Council to be notified of its right to make such appointments, and that any proceedings of the Commission taking place after thirty days from the date of such notification shall be valid, although all or any of such appointments shall not have been made.

2. *Duties.*—It shall be the duty of such Commission—(*a*) to carry out, so far as in its power lies, such instructions as the Congress shall give to it Session by Session; (*b*) to note the course of medico-political events of importance in Australasia, and, where, in its opinion, intervention by it, as representative of the medical profession in Australasia, is urgently called for, to take such action as it may deem appropriate to safeguard the interests of the profession; (*c*) to be at the service, as an advisory body, in medico-political matters, of the Government of the Commonwealth of Australia and the Dominion of New Zealand; (*d*) pending the adoption, by the Congress, of rules for the conduct of its proceedings, to make such rules as it may consider expedient for this purpose. Provided that until the making of such rules, and in any matter not provided for in such rules when made, procedure shall be determined by the President. Provided also that at any meeting of the Commission four shall be a quorum; (*e*) to meet from time to time at such places and times as the Commission, or, failing the Commission, the President may fix; (*f*) to offer from time to time such reports of its proceedings, as the President may approve, to the *Australasian Medical Gazette*, the *Australian Medical Journal*, and such other medical newspapers as the Commission may select, for publication therein; (*g*) in the event of any officer appointed by the Congress dying or ceasing to be able to act while the Congress is not in Session, to fill the vacancy thereby occasioned; (*h*) to report to the Congress at each Session thereof in regard to its proceedings since the previous Session.

3. *Powers.*—Such Commission shall have power—(*a*) pending the adoption by the Congress of rules for the conduct of its proceedings, to alter, amend, or rescind any rule made by it for such purpose; (*b*) to delegate to committees, consisting of one or more of its members, such particular duties and powers as it may specify in any resolution appointing any such Committee, and any such Committee shall have power, with the express approval of the Commission or the President, to cooperate with any member of the profession specially qualified to assist it—provided that no such Committee shall continue for more than three months unless a longer period be fixed by the Commission; (*c*) to form sub-committees of its members with such powers and duties as may be deemed by it to be expedient.
4. *Expenses of the Commission of Congress.*—That surplus moneys in the hand of the Honorary Treasurer, after the expenses of each Session have been met, shall be at the disposal of the President to meet the expenses of the Commission of Congress.

Dr. Bowman seconded.

Dr. Hayward: Members of different branches perhaps know that they very kindly decided to send delegates to consider the suggestion which I have brought before the British Medical Association in connection with an autonomous medical association here. I think the present meeting is too small to consider such an important and serious question like this. It must not be dealt with offhand. We cannot do it justice while everyone is rushing away to get somewhere else, and I am going to propose that the subject be put to the following committee:—Drs. H. L. Maitland, W. H. Crago, R. H. Todd, J. A. Dick, R. D. Fetherston, G. A. Syme, R. R. Stawell, F. S. Hone, W. T. Hayward, H. S. Newland, Gregory Sprott, G. W. Harty, W. W. Love, J. S. C. Elkington, W. N. Robertson.

This represents influential members of each of the States. They can consider the matters that may be brought before you, and at the same time consider this other question which has been mentioned by Dr. J. Adam Dick.

Dr. David Thomas: I have much pleasure in seconding this. It would, indeed, be a very great pity that such a very important subject like this should be discussed at the tail-end of the meeting, when everybody is rushing away. This is a question which affects the whole profession considerably, and I think the time is not opportune here to consider such a very important matter.

Dr. R. H. Todd: In regard to this motion in Dr. Dick's name I formally approve of it, and I look forward to the time when there will be, between the meetings of the Congress, an organised body of sufficient authority to deal with all matters of medico-political interest, which have been referred to it by this Congress. The want of such a body has been felt, and in the report of the Executive Committee, which has been put before the meeting to-day, this matter was referred to. It was on the existence of that paragraph referring to this matter that the idea came that a Standing Committee should be appointed, having certain powers and duties. Our friend, Dr. Hayward, has suggested that this matter be submitted to a committee, and he has named those who should form that committee. I think, as Dr. Thomas said, such a matter should not be passed over lightly, or dealt with by a very small meeting, and even if dealt with by a large meeting, I think it would be better if we referred it to a committee to deal with it again at this Congress at a subsequent meeting. It must be clearly understood that time should be given at the final meeting to deal with this matter. It should not be shelved. We ought to have some guarantee of that. I am sure that what the President can do in this matter he will do, and I am sure he will see that it is not lightly passed over.

In regard to the committee that Dr. Hayward has proposed, the names he mentions are delegates appointed by the different States to consider some other subject not connected with the Congress at all. I think, myself, that they are very representative men, and that, as such, it would be very good to appoint them as a committee of this Congress, it being understood, of course, that this is an entirely separate matter. I have very much pleasure in supporting the amendment.

PRESIDENT: Of course I cannot guarantee that the meeting on Saturday morning will be any better attended than this; it will probably be worse. I quite agree it is a matter requiring a great deal of time and care, and I think you may rely that every attention will be given to it.

The amendment was put to the meeting and carried unanimously.

PRESIDENT: As Dr. Purdy is not here, and as he has made no arrangement for anybody to propose the resolution standing in his name, it must lapse.

The proceedings then terminated.

INAUGURAL MEETING.

HELD AT

TOWN HALL, SYDNEY.

MONDAY EVENING, 18TH SEPTEMBER, 1911, at 8.30 p.m.

On the dais were their Excellencies Lord Denman (Governor-General), Sir William P. Cullen (Lieutenant-Governor), Vice-Admiral Sir George King-Hall (Naval Commander-in-Chief), Archbishop Wright (Primate of Australia), Archbishop Kelly, Sir Allen Taylor (Lord Mayor), Sir Francis Suttor (President of the Legislative Council), the Hon. J. S. T. McGowen, M.L.A. (Premier of New South Wales), the Hon. Josiah Thomas, M.H.R. (Postmaster-General of Australia), the Hon. J. L. Treflé, M.L.A. (Minister for Agriculture), the Hon. W. A. Holman, M.L.A. (Attorney-General), the Hon. Sir Normand MacLaurin, M.D. (Chancellor of the University), Sir Philip Sydney Jones (past President of Congress), the Hon Dr. G. H. Butler (Representative of Government of Tasmania and past President of Congress), Professor Allen (past President of Congress), Professor Anderson Stuart (Dean of the Faculty of Medicine), Dr. Ashburton Thompson (President of the Board of Health), Dr. B. B. Ham (Representative of Government of Victoria), Dr. A. A. Lendon (Representative of Government of South Australia), Dr. James Hope (Representative of Government of West Australia), Dr. Arthur Palmer (Honorary General Secretary of Congress), Dr. S. H. MacCulloch (Hon. Treasurer), Dr. Brady (Associate Hon. Secretary), Captain Stewart Richardson, Captain Verney, Flag-Lieutenant Seton.

In the Reserve beneath the platform were Her Excellency Lady Denman, Lady Cullen, Sir Walter and Lady Barttelot, Mrs. McGowen, Miss Quirk, Mrs. Antill Pockley, Mrs. Palmer, Mrs. MacCulloch, Mrs. Brady, Mrs. W. G. Armstrong, Mrs. Bowker, Major Quilter, and in the body of the hall were a large number of members of Parliament, Judges of the Supreme and District Courts, Members of the Senate of the University, and other leading citizens, including a large number of ladies, in addition to members of Congress and their wives. The Eastern gallery was filled with nurses from the Sydney and Suburban Hospitals in uniform, and a number of medical students were also present.

The meeting was preceded by a Recital on the Great Organ by the City Organist, Mr. Truman, from 7.30 to 8.30.

Dr. F. ANTILL POCKLEY (President of Congress) occupied the Chair.

PRESIDENT: Your Excellency, it is my honour and privilege, on behalf of the Congress, to tender to you and to Her Excellency, Lady Denman, a respectful and cordial welcome to our Inaugural Meeting, and to ask you, Sir, to honour us by opening the Congress.

LORD DENMAN (who was received with great acclamation) said: Mr. President, Ladies, and Gentlemen,—I thank you for the very kindly reception which you have just given me. I can assure you that I esteem it a very high honour to have been asked to preside at this meeting of the Australasian Medical Congress. Though but a recent arrival, I think my first and obvious duty is to extend a hearty welcome to the delegates and members, more especially to those who have come from other States, and from other parts of Australasia; and I am sure that this city is proud that the Ninth Session of the Australasian Medical Congress is being held within its boundaries. This triennial meeting is unusually important, because not only are there many visitors from other States, but there are many visitors also from New Zealand, and I believe that this Congress is being more largely attended than has ever been the case since its inception.

I should naturally hold it a compliment to be asked to attend, or to preside at a gathering representative of any of the great professions, but the medical profession has a special claim to be regarded as a branch of the public service. It is true that physicians and doctors do not always endear themselves to mankind. Occasionally we are not grateful, perhaps, to those who inflict pain and privation upon us, no doubt purely for our own good, and sometimes we think they are rather harsh and unnecessarily severe towards our pet weaknesses; but, speaking generally, society is grateful to the medical profession, and it puts forward a claim upon their services which it would not prefer against the members of any other profession that exists. It imposes upon them a high and disinterested standard of duty, and it exacts from them gratuitous work and personal self-sacrifice in the cause of humanity.

Yesterday I had the privilege of listening to an eloquent sermon from the Primate of Australia (who I am glad to see on this platform to-night), who claimed the co-operation of the medical profession with the Church. I would further maintain that an alliance should and does exist between the medical profession and the State. Everywhere there is increasing recognition of the importance of the study of public health as a part of the science of government. In England the trend of modern legislation to-day almost puts the medical practitioner in the place of the public official. In this Congress, I note that the subject of public health and State medicine is to be introduced by a delegate from Tasmania, and discussion will also be initiated on medicine and surgery, as they affect naval and military services, by Colonel Purdy of New Zealand. But even more technical and apparently abstruse branches of medical science have almost a direct bearing upon the public welfare. Scientific research in the laboratory is a necessary preliminary to the saving of life and of the alleviation of pain and suffering in the hospital and in the home. Men who devote their lives to investigations of this kind are literally crusaders in the cause of humanity, for they give singly, unselfishly, and disinterestedly their studies to carrying on warfare, and strenuous warfare, against disease on behalf of the human race. (Hear, hear.)

Now, ladies and gentlemen, there is a fairly long agenda paper this evening, so I will not detain you longer. I will only say that I have much pleasure in declaring this, the Ninth Session of the Australasian Medical Congress, duly open. (Loud applause.) I will now ask the Premier of New South Wales, Mr. McGowen, whom I recently had the pleasure of meeting in England, to address the meeting.

Mr. McGowen (who was received with applause) said: Mr. President, Ladies, and Gentlemen,—A little girl one time said to her mother, "Mother, can I say my prayers in bed, like Mary?" "Why? Does Mary say her prayers in bed?" asked her mother. "Yes," said the little one, "as soon as the man knocks at the door in the morning Mary says "Oh, God, I have got to get up." Just like Mary, I feel on this occasion that I wish I had not to get up, and yet again I feel that I have something to say, and I want to say it to the members of this Congress on behalf of the people of the city of Sydney, and the whole of New South Wales.

I am one of those who agree with His Excellency the Governor-General, that the medical profession is the noblest profession that we have in our civilisation; it has given us more princes among men than any other profession; it possesses in its ranks, in every civilised country, men who unselfishly give their learning and their research willingly and freely for the benefit of suffering humanity. When we realise in our lifetime what has been done by men who have been martyrs to their profession, we know that there is no monument, not even the monument in Trafalgar Square, that can bear testimony of what suffering humanity owes to the medical profession. I glory in the fact, Sir, that when these men, by their unselfish effort, by their burning of the midnight oil, make a discovery, they offer it to the world; it knows no country, it knows no colour. Let a discovery be made in the East, or in the West, on the Continent, or in Australia, it is sent all over the civilised world to benefit suffering humanity. And I, who, as you know, come from the working classes of this community, know that if there is a profession that is considered noble, if there is a profession for which working men have a veneration, it is the medical profession. They honour the men who render them such great service. Other professions, when they make a discovery, look after the "patent rights," but the medical man has no thought but for the benefit of suffering humanity. To-day, in our lifetime, we see that pain has been alleviated, that lives have been saved by the discoveries of these self-sacrificing men in the laboratories to which His Excellency has referred. Just one other word. Travel in the interior of these new countries like Australia, and you will find perhaps unknown—comparatively speaking, unknown—modern scientific improvements in relation to hospitals. You will see the medical man and the ministering angel in the country hospital alleviating pain and saving life; and as His Excellency the Governor-General states, the legislator looks to the medical man to tell him what is the latest discovery with regard to the food we eat, and the conditions under which our people live. We have to bear our testimony as laymen, that individual life has been lengthened, that comforts have been increased, and the sufferings of mankind alleviated. I bid the Congress welcome to this sunny country in the south. May the sun shine, figuratively and literally, on its deliberations, and may humanity reap a further benefit from the unselfish devotion of the members of the medical profession. (Loud applause.)

Sir ALLEN TAYLOR (Lord Mayor of Sydney): Your Excellency, Mr. President, Your Grace, Ladies, and Gentlemen,—I am exceedingly pleased to have the privilege of confirming the sentiments expressed by the Premier of the country, and to welcome the members of the Congress.

I am sure, from the utterances of His Excellency the Governor-General to-night, it is evident to everybody that the humblest individual in this great Commonwealth realises the importance of this Congress, which has now entered upon its labours. The Premier has been good enough, on behalf of this State, to offer each and every member of the Congress a real, true, and warm hearty welcome to this State. (Applause.) Therefore, Your Excellency, as the Mayor of this great city, it is more than a pleasure on my part to endeavour, to the best of my judgment and ability, to confirm the sentiments of the Premier. But may I say this: that I look upon this great conference as one of the most important events that has taken place in the Commonwealth in the last twenty years, and I am told that it will be nearly twenty years before we will have the privilege of once more extending a welcome to the existing

President or those who may follow that distinguished gentleman. The President of this great movement has every reason to be congratulated, and more than satisfied, with the prospects of the great Congress which we have opened to-day. We fully realise that he has done an enormous amount of work to bring this great movement to its present position, and the members of the Congress have rendered their distinguished chief every possible help to make this movement worthy of success. We have every reason to believe that the results achieved, as outlined by His Excellency, will be far-reaching, and the outcome of that great labour will, as has already been indicated, be provocative of good to the community as a whole, as well as to the medical profession. Whatever the benefits of this Congress are, rest assured that sufficient seed will be sown for mature thought; and it is only a matter of time when an enormous amount of good must come from this movement.

Your Excellency, I have very much pleasure indeed to be associated with this movement, and I wish to give each and every delegate, together with the President, a real and true welcome to the shores of this State, which everybody appreciates has enormous possibilities.

PRESIDENT'S ADDRESS.

PRESIDENT: Your Excellency, Ladies, and Gentlemen,—It is my next privilege to welcome, on behalf of Congress, their Excellencies the Lieutenant-Governor, the Naval Commander-in-Chief, their Graces the Archbishops, the representatives of the Legislature, the Premier, the Lord Mayor, the Chancellor of the University, and all those ladies and gentlemen in my audience not belonging to our profession, who, by honouring us at a gathering which is not in the nature of a light entertainment, are showing their goodwill and their interest in our proceedings. On behalf of the profession in New South Wales, I wish to extend a hearty welcome to our brethren from beyond the State, and to express the hope and belief that our discussions and intercourse will be to our mutual benefit and to the advancement of our knowledge. We hope, also, that our visitors will enjoy the social relaxations which form an important and not unprofitable part of the proceedings of Congresses, and that when they leave us they will carry with them pleasant memories of their visit to the mother State, and of the charms of this fair city and its surroundings.

I would also express pleasure at seeing so many of those who have been prominent in the work of former Congresses; amongst them it is good to see with us, in full physical and intellectual vigour, my distinguished predecessor in this Chair at the last Sydney Congress, Sir Philip Sydney Jones. (Applause.)

There is, however, one well-remembered face that we miss, that of the late Dr. John Thomson, formerly doyen of the profession in Queensland, and President of the Brisbane Congress in 1899. His striking personality, genial bonhomie and kindly hospitality cannot be forgotten by those who had the good fortune to meet him. Very soon after the last Congress in Melbourne, where he was, as usual, a conspicuous figure, he was stricken, and died. The resolution of condolence passed at the meeting this morning will, I need hardly say, be sympathetically endorsed by every member of the Conference.

We have to regret also, that Sir Frederick Treves, Sargeant surgeon to no less than three British sovereigns, the accredited representative of the British Government to our Congress, has been prevented by illness from attending.

In offering my thanks to Congress for having unanimously elected me to the presidentship, I am discharging no vain formality. Though never in my most ambitious moments aspiring to such an honour, one that can fall to the lot of only a few men in a generation, I esteem it as the very highest that any man can receive at the hands of his fellows, denoting as it does the possession of their trust and confidence and respect. Though more than dubious of the wisdom of your choice, I have no misgivings as to your goodwill. Being under no delusion as to my own limitations, which are too obvious to be covered by any apology, and having in mind the pre-eminence of my predecessors in this Chair, it is with much humility that I approach the task of giving an address worthy of the occasion.

As it is my lot to be a worker in the ranks of the profession, and in one special branch only, I comfort myself with the thought that the many among you, who, like myself, are everyday practitioners, will realise my position, and not expect a learned dissertation or authoritative statements on the many profound and intricate problems that are exercising thought in the medical world to-day. Custom, and not my will, prescribes a presidential address. For my disabilities I crave your indulgence. (Loud and continued applause.)

The nineteen years that have passed since Congress last met in Sydney have been marked by great progress and evolution in every previously recognised branch of medicine, and by the discovery and opening up of quite new fields.

We have seen, for instance, microbiology and biochemistry, then in their infancy, developed and elevated to the dignity of sciences, and, along with fuller knowledge in the problems of heredity and immunity (at that former time only dimly and vaguely apprehended), changing our ideas as to the causation and processes of disease, and modifying our therapeusis; and we have seen the birth of such new and strange things as radiology and parasitology.

More universally and rapidly than at any time in its history, medicine is passing from the traditional and empiric, and becoming more rational and scientific. New vistas are being opened up through the jungle of mystery and ignorance. From many new sources illumining rays are reaching out into the darkness; but, while dispelling some of the obscurity immediately around us, and throwing fitful beams on more distant spaces, they at the same time reveal the density of the encompassing darkness and enlarge the periphery of our ignorance.

It is difficult to survey a position that is changing with such bewildering rapidity that we scarcely know where we stand; when even those who are devoting all their time and intellect to the elucidation of even one of the many medical problems of the day are scarcely able to keep up with the prolific production of literature on the subject.

The discoveries already made, and others foreshadowed, are threatening not only to revolutionise the practice of medicine, but, within limits which inexorable Nature ever ordains, to profoundly alter the inter-racial relationships of man and influence his distribution on the face of the globe.

It is mainly through knowledge of the causes and processes of microbic disease, and of the way to prevent, or to imitate, modify and control the action of bacterial and protozoal organisms and their products, that these results will be brought about, and by studying Nature's methods of conferring immunity and trying to use these methods in a gentler way. To this end, biologists and chemico-pathologists are diligently and strenuously working, experimenting, and investigating throughout the world.

But the difficulties are great and baffling. Even in regard to bacterial or protozoal disease, where we have a known or assumed specific cause, the problems to be solved are of extraordinary complexity. We must all be impressed by the magnitude of the task of these scientists, by the enormous amount of work done in a few years, by the results already attained, and the accumulated stores of information, as well as by the brilliancy of intellect, the powers of observation, the acuteness of perception, the marvellous patience and perseverance, the wonderful ingenuity displayed in the experiments, and the cleverness and beauty of the theories evolved.

To the debt the whole world lies under to that immortal son of France —the country that has given so many noble servants to science—Louis Pasteur, the man who "raised the veil that from the beginning of time had covered zymotic disease, discovered and demonstrated its microbial nature," and incidentally inspired Lister—to that debt compound interest is mounting up to those who have carried on the torch lighted by Pasteur, disciples of his—many of them his immediate pupils—Eli Metchnikoff, Koch, Ehrlich, Laveran, Yersin, Wright, Manson, Ross, Reid, Carroll, Lazear, Von Behring, Kitasato, and hosts of others, men of every country, including an illustrious representative of our own in Bruce, the hero of Malta fever and sleeping sickness.

On immunity and specific therapy I am not qualified to speak, except in the most general terms. It is, however, in the elucidation of these problems that we have our best hope of success in coping with microbial disease. And yet we feel we are only on the threshold, dimly guessing at the stores within. The door is often rudely shut in our faces, and we have to laboriously seek another entrance. The little knowledge we have is not yet filtered, but is swarming with germs of ignorance and error. The field for exploration is immense, some of it yet untouched. We are like the child with the pebble on the beach, and may well feel with Tennyson—

> So many worlds, so much to do,
> So little done, such things to be.

More than at any former time, the man who would keep up with the march of medicine must avail himself in his daily work of the help given him by the scientist in his laboratory. The insistence of the biochemist, the biologist, and the physicist is tending indeed to make us medical scientists, rather than practitioners, relying chiefly on our trained faculties and powers of observation and clinical experience.

Not yet, however, has the new displaced or eclipsed the old. We do not find the living body respond as regularly and consistently to purely scientific methods as does the test tube of the chemist. There are elements and energies and processes in the complex living body of which we are as yet profoundly ignorant, that modify or alter, or upset laboratory results. In many cases there are inherent and, in the present state of our knowledge (or ignorance), insurmountable obstacles to these methods being even tried in the human body. Even such laboratory diagnostic methods as examination of the blood, whether serological, cytological, or chemical, are by no means always decisive. Though sometimes confirming the diagnosis of the clinician, they frequently serve but to increase his difficulties. The complement deviation test of Wassermann is an example.

Results obtained in vitro in sterile media of known composition, under accurately controllable conditions, as to proportions, time, temperature, &c., are by no means always in accord with those brought about in the living body, of which we know neither the composition nor the energies, and cannot control the workings. It is in the living body that the fight against disease is to be waged, not in the laboratory test tube, and no two bodies, probably no two cells, are precisely alike, nor do we know in what the differences lie, nor shall we know as long as we cannot answer the question, " What is protoplasm?"—with which is correlated the still deeper mystery, " What is life?"

We can only say that the boundary between life and "not life" does not appear to be so fine as has been supposed, and we even find the Cartesian doubt of the scientist again questioning the proof of the doctrine, *Omne vivum ex vivo*, with which Pasteur appeared to have finally settled the controversy with the heterogenists. Apart from the wonder of osmotic growth, we have men like Bastian claiming that living matter is capable of coming into existence.

We can say, at least, that many important facts have been discovered, underlying principles, at least perceived, if not comprehended, and working hypotheses formulated. We must, however, avoid the temptation to be lured on to the quicksands of speculation, but keep to the firm ground of proved scientific and clinical facts. With a multitude of investigators, many apt perhaps to be carried away by their enthusiasms and aspirations, we are now, more than when Huxley uttered it, in need of keeping in mind his warning of the danger of deduction and generalisation about the facts of biology. The methods of science have to be applied to our reasoning and interpretations just as rigidly as to our experiments and their results. In the words of Clifford Allbut, " These ways are strewn with conjecture and speculation, withering untimely even before the early spring of a working hypothesis."

We have evidence that man is naturally immune to many diseases to which other animals are susceptible. We believe that some individuals are naturally immune to certain diseases that affect others. We also believe that, apart from natural inborn immunity, man, speaking generally, acquires an immunity to any particular microbic disease by having successfully passed through an attack of that disease, the immunity in the case of some diseases being, as a rule, lifelong and absolute, whilst in others it is of very short duration—a few weeks or less. We suppose that in no other way than by acquiring immunity against the specific organisms or their products does he recover from the disease.

Attacks of zymotic disease cause the body cells and fluids to acquire new characters, and these new characters are transmitted to the descendants of the cells, and so acquired immunity results. Probably the process by which the cell attains immunity is one of natural selection and variation, and not a use acquirement; but this is not yet established. The life and death struggle between the body cells and the invading organisms may be at close grips between the phagocytes and the microbes, or at long range between the fixative and digestive secretions of the defensive cells and the toxins of the invading germs. Whichever it be, there is a struggle for survival between the contestants on either side, and it would appear that the microbes on their part are capable of developing a counter immunity against the natural or artificial forces brought against them. We see this in the gradually increasing resistance that the parasites of malaria and sleeping sickness offer respectively to quinine and arsenic. Moreover, Ehrlich has been able to cultivate a strain of trypanosomes with an immunity to certain poisons—fuchsin and arsenic—not possessed by the parent parasite.

It is the common belief that every microbic disease has but one specific germ as its cause, and that where the same disease, or a pathological condition clinically identical is produced by another germ, or another cause, it must be regarded as a pseudo or false disease. Some, however, regard this as a generalisation not proven, and recent observations, in typhoid fever, for instance, seem to throw doubt on the bacillus typhosus being—as has been hitherto held—the sole etiological agent. The frequent negative results of Widal's test in clinically undoubted typhoid, and the finding of a positive Widal in diseases clinically not typhoid, lend support to this doubt.

The fact that persons can carry about with them for long periods, even years, billions of the supposed causative bacilli of typhoid fever, diphtheria, and other diseases, without ever showing symptoms of the disease, has not yet been satisfactorily explained. The problem for the scientists to solve is the nature of the processes by which the system reacts against the invading organisms, and to find means of assisting the individual in these processes, whether it be by fortifying the phagocytes, the production of antibodies, alexins, opsonins, antitoxins, or what not, or by supplying the system with the antibody already formed.

Although already a considerable measure of success has been attained in several diseases, the results, so far, fall short of our expectations. If the problem be such an extremely difficult one in the case of microbic disease, of which we believe we know the origin, and can isolate and cultivate the germs, and even cultivate different strains (as a farmer breeds different strains of sheep), can isolate their toxins, increase or diminish their virulence, and experiment with the microbes or their products in various ways, how much more are we in the dark in regard to the processes of immunity in the non-microbic diseases—such as the power of resisting poisons, adverse physical conditions, and metabolic disease generally, where we have little or no knowledge of their initial causes, and scanty information as to the physiological processes or pathological disturbances of the economy that occur.

The task of one who sets himself to investigate the metabolic processes at work in the animal organism, and the derangements which these processes undergo in disease, has been likened to that which confronts a man who wishes to study the manifold activities of a great chemical factory which he is forbidden to enter. He is able to examine the raw materials which are brought to the factory, the finished products which emerge from it, and the waste substances which are washed away in the drains, escape from the chimneys, or are strewn upon the dust heap; upon such examinations, supplemented by an occasional peep through a window, he must rely for his information as to what goes on inside (Garrod). And yet, with so little means at their disposal, chemical physiologists and pathologists have found out much, and are, year by year, making fresh incursions into the vast territory of the unknown.

The discoveries already made in medical science are more vivid, more striking, more dramatic, if one may use the word, more easily formulated and put into words, and more quickly assimilated than are the patiently accumulated stores of clinical experience. They are apt to appear out of their true perspective to the practitioner of the younger generation, and to appeal to him as a short cut to a knowledge of the processes of diseases, and the way to cure it.

The present position would appear to be that while we get valuable help in diagnosis, as in tuberculosis for example, and in treatment, notably in diphtheria and in a few other diseases, from the biologist, yet he is only ancillary to the clinician. The sound practitioner of long experience and ripe judgment, with his trained eye and ear and touch and smell, and, even in some cases, taste, but acquainted with and ready to take advantage of all proved scientific aids, remains—and probably for long will remain—the man to whom we instinctively turn in all grave crises in our clinical work.

The increasing scope and complexities of medicine are adding to the burden of the already overtaxed medical student, who not infrequently breaks down under the strain. In thirty years the course of study has been lengthened from three years to five, and the longer period is now altogether insufficient.

Although the minimum is five years, the mean length of the curriculum in Great Britain and in Australasia is a little under seven years, and the average age at graduation about 26.

This is not the place to discuss the various proposals for relief, but it is plain something will have to be done.

One will be expected to say a word about one notable and quite recent discovery, which has caused quite a stir in the medical world, and is being hailed with joy and satisfaction by a very large proportion of mankind. I allude to that by the great synthetist, Ehrlich, of a supposed cure for one of the most serious and loathsome and most universally distributed disease that has ever affected the human race. The remedy is neither a serum or a vaccine, but a carefully thought-out chemical combination—di-oxyamino-benzol di-chloride—also known as arseno-benzol and other names. One of these, "606," represents the number in the series of the combinations experimented with, and shows the amount of patient investigation carried on before the present compound was adopted. Though neither a serum nor a vaccine, it is claimed that serum from a patient injected with arseno-benzol and milk from an injected mother has similar immunising properties to the drug itself. If its claims are sustained, this drug will be noteworthy as being the only experimental one known with the property of rapidly destroying an infective organism in the blood without injuring the host; the only other, indeed, that has this power is quinine, which destroys the organism of malaria (like the other, a protozoon). The use of this alkaloid, however, was empiric, and it was made known to us by untutored savages. Ehrlich's remedy was the result of careful scientific research and deduction based on the previously ascertained fact that certain compounds of arsenic had been found to be poisonous to certain protozoa, *e.g.*, trypanosomes. These other preparations, however, such as atoxyl, have been found to produce serious and even fatal toxic effects on the individual, which is said not to be the case with arseno-benzol.

While it has produced good results in spirochœtal disease, whether it will permanently cure it, or be more beneficial than former methods, is as yet uncertain. All we say is that it is on trial.

Meanwhile, we have a note of warning from the veteran, Jonathan Hutchinson, as to the possible risk of arsenic originating or intensifying a proclivity to the growth of cancer.

I do not propose to spend time in the enumeration of modern surgical triumphs. The ground may be covered by the statement that with the exception of some parts of the brain and spinal cord there are no parts of the body sacred from the surgeon's knife.

Up to 1909 there were 150 recorded operations on the heart itself, with 35 per cent. of the patients saved from an otherwise certain death.

The President of the Royal College of Surgeons said recently that surgical operations are performed so well and so frequently that the public has come to regard them as a form of innocent recreation—whether for the patient or for the surgeon, he did not say.

It is only within the last twenty years that the vermiform appendix has acquired its evil reputation. Now appendicectomy is so common and, one might say, so fashionable, that a person who has not had the appendix removed is considered hardly respectable.

A little more than twenty years ago it was considered almost criminal to open the abdomen, and nearly all acute inflammations of the abdominal cavity were fatal, death being due to peritonitis, generally *beginning* in appendicitis.

The most notable of the recent advances are probably the extension of brain surgery, and transplantation operations, one of the latest of these being transplantation of nerves, with restoration of function to paralysed muscles; and another, transplantation of a length of intestine to make a new gullet in place of one destroyed by injury or disease.

Whole organs have been successfully transplanted in animals, and ere long we may expect to see this done in man, though it is not clear where the supply of spare parts is to come from.

We have not yet arrived at such advertisements as the following, attributed by an American humourist to the surgeon who had destroyed his conscience:—"Medical Colleges and Hospitals desiring assorted tramps for scientific purposes, by the gross, cord measurement, or ton, will do well to inspect the lot in our cellars before deciding elsewhere."

Most of the advances in surgery have been made practicable by first experimenting on animals, a procedure so foolishly anathematised by a certain well-meaning but misguided section of the public, who are provided with employment or outlet for their superfluous energies in running the really antihumanitarian cult of antivivisection. I wonder whether, if put to the test, an antivivisectionist mother would refuse to her child, smitten with diphtheria, the practically certain cure by the injection of antitoxin, though she knew that the serum was got by vivisecting the horse, the noblest of animals. Would she not rather go on her knees and thank God for this life-saving medium? We have had local experience of Christian Scientists refusing such aid, and death resulting; but what better can be expected of people who say neither microbes nor disease have any existence. But this is a digression.

When dealing with such a marvellous form of energy as radio-activity, as was to be expected, the temptation to make exaggerated claims for its usefulness in medicine has been irresistible, especially to pseudo scientific writers in the lay press. An infinite amount of harm has been done by this. False hopes have been raised in the minds of sufferers from various diseases, and, while waiting for radium, many have put off till too late the operation which alone could have saved them. We have seen a repetition of the irresponsible writing (so deprecated by Koch himself) that gave forth premature and unjustifiable statements of the curability of consumption by tuberculin, in consequence of which all roads to Berlin were dotted with dead and dying victims of this fell disease.

There is a growing belief that nearly all, if not all, the therapeutic effects of radium can be got by the X-rays, and at much less expense, considering the fantastic price of radium, and even of other comparatively cheap radio-active substances, such as mesothorium (which for medical purposes is apparently quite as useful as radium). Indeed, the gamma rays of radium are considered to be identical with X-rays. Amongst the claims for its almost universal therapeutic value it is almost with a feeling of surprise that we find the admission that radium emanations do not affect bacteriodal, antitoxic, and other biological actions, and do not cure real cancer (carcinoma).

The conservative scepticism of surgeons in withholding judgment is being justified. While sometimes small rodent ulcers, and small epitheliomata, and superficial sarcomata may be apparently cured by it (as by X-rays), it appears that it cannot cure, or even benefit real cancer; sometimes does more harm than good, and cannot prevent spreading of the disease, recurrence, or metastasis. Early excision of the growth by the surgeon still remains the best hope for the patient.

Although many men are devoting their lives to the elucidation of the intricate and tantalising problems of the nature and origin of cancer, so far no definite conclusions have been reached, but we are not without hope that the pieces of the puzzle may be fitted together, and the meaning of it all made clear. Whether cancer be bacterial or protozoal in origin, or whether arising from normal cells run riot, we do not, with certainty, know, though evidence against its being due to micro-organisms is accumulating. We have learnt that cancer occurs not only in man, but that all vertebrate animals are liable to a disease indistinguishable from cancer, though whether this be true cancer or not is still controversial. If true, it is strong evidence against the bacterial or parasitic theory, for it is against all experience for any one micro-organism to attack all vertebrates.

Cancer can be grafted from animal to animal (though with more difficulty than in the case of microbic disease), but apparently it cannot be grafted from one animal to another of a different species. In this specificity we see the working of a biological law.

Though we know little about its intracellular metabolism, it would appear that it possesses certain chemical peculiarities, but develops no toxin. It has also been demonstrated that the cancer cell often shows symmetric division, and contains only half the number of chromosomes of a body cell, thus resembling a gamete. The significance of this fact has recently had some light thrown upon it by some noteworthy researches by Ross and Cropper, in cell reproduction. Whatever may be the outcome of these investigations, our hope of a means of curing cancer, other than by the crude method of removing the growth, lies mainly in the belief, justified by experiments on animals, and by clinical experience, that there is some form of immunity to cancer in the individual ; and in our knowledge that undoubted cases of cancer have been known to disappear without any treatment whatever. There must be, at times, something in the body, or in the conditions governing cell proliferation, that is inimical to cancer. If this be so, we have a reasonable hope that this condition, or property, or substance may be found, and then we may be able to imitate the natural method of cure.

At present, for true cancer (carcinoma) early removal by the surgeon is the only rational method of treatment, and the more widely this is known the better for all concerned. A minute focus, no larger than a pin's head, may be a fully developed cancer with all the properties of malignancy. The appreciation of this fact has revolutionised the treatment of cancer, as in the tongue, for example. We do not wait and watch the development of a suspicious growth, ever so minute, but insist on immediate excision. Removal by caustics, favoured by quacks, is a slower, more painful, more dangerous, and less effective method of attempting to destroy it. X-rays and radium have a limited application, and only in some forms of malignant growth. They have proved useless and even dangerous when applied to true carcinoma.

No known drug, no patent nostrums, not even the locally-vaunted Mackay molasses treatment, are of any value, but only dangerous, inasmuch as valuable time is lost while they are being tried. Cancer is a circumscribed and not a constitutional or blood disease, though we have to recognise certain kinds of predisposition in the individual, in age (in which incidence cancer is unique), in sex, in race, and in the coexistence of certain conditions, such as alcoholism and enthetic disease, injuries or chronic irritation, and we also know that certain organs of the body are more liable to cancer than others.

The stigma for long attached to medicine, that its advance had not kept pace with that of surgery, has in recent years been removed. It is mainly in prevention that medicine has achieved its greatest victories, but in many other directions it is progressing, notably in organo-therapy, vaccine, and serum therapy, and in the treatment of pulmonary, cardiac, digestive, and enthetic diseases.

We hear a great deal nowadays about the "romance of medicine," and to nothing in its history is the character of "romance" more applicable than to the story of malaria, yellow fever, sleeping sickness, and other parasitic diseases. This interesting and wonderful story has so oft been told, and, along with the unparalleled record of the success of preventive measures, is so well known to all the reading public, that, alluring though the subject is, I shall not touch upon it here.

The inhabitants of the tropical and sub-tropical parts of Australia suffer from many insect-borne diseases, and practically nothing has been done in the way of prevention. There is a fear in the minds of some that the dreaded yellow fever may reach our shores through the opening of the Panama Canal. The danger is not altogether imaginary. We have the transmitting mosquito (*Stegomyia fasciata*, or *calopus*) already in Queensland, and it has been found to the south of Sydney, though not in Sydney itself.

If the mosquito sucks the blood of an infected person, there is a latent period of twelve days before the insect becomes infective and capable of transmitting the disease to man, and a further incubation period of five days before a man so bitten would show the disease, and the voyage from Panama to Australia might be accomplished in less than seventeen days. Our probable safety, however, lies in the fact that yellow fever and the stegomyia have been practically banished from the Canal zone, and that, according to Colonel Gorgas, of the United States Army Medical Service, the most experienced authority on the subject, it requires ten stegomyiæ to the square yard for yellow fever to spread.

We have seen the successful resistance for centuries to the occupation, by alien races, of the West Indies, Central America, parts of Africa, and other tropical countries, by malaria and yellow fever, which slew the invaders by millions. Now we see how the conquest of the mosquito is making these same countries more habitable by the white races.

It is claimed that by the prevention of these and other parasitic diseases we are opening up vast zones to commerce, and to occupation by the white races. Though this be true, yet the ultimate results may be far different. It must be remembered that the indigenous races have not an absolute, but only a relative, immunity to protozoal diseases, through a long process of selective adaptation. From the prevention of these diseases the dark races will also benefit, and their far greater fertility as compared with that of the whites will result in their relatively far greater multiplication. Again, the constitutional laziness of the negro is attributed to a very large extent to the hookworm and its resulting anæmia. With the prevention of protozoal diseases, therefore, we must expect the dark races to show an increase, not only in numbers, but also in energy and capacity and inclination for work, and so to become more formidable competitors than heretofore for the possession of their ancestral domains.

But, apart altogether from the effect of parasitic diseases, it is questionable if the white races can ever permanently occupy the tropics.

The existing divisions of mankind are the outcome of their special environments. They are what climate, soil, diet, heredity, and natural selection, acting by very slow stages, through tens of thousands of years, have made them. Rapid migrations are fatal. If a new zone differs but little in character from the old, the variety persists and adapts itself; if not, it either becomes absorbed in the indigenous element or else dies out (Read).

Apart from parasitic disease, the factor that of all others militates against the white man in the tropics, is the effect of the actinic light of the sun, the violet and ultra violet rays, against which the negro is protected by the pigment in his skin and eye.

These short ether waves have a most disturbing effect on protoplasm, which, like all compounds composed largely of nitrogen, is very unstable. They act in two ways—first, by stimulating the metabolism, and, secondly, by deranging the molecule and destroying the protoplasm.

Investigations in the Philippines have shown further that brunettes do not deteriorate to the same extent as blondes; and Ripley gives figures to show that in the tropics the death-rate of alien races is in proportion to the distance they are removed from their ancestral zones.

These and many other facts, which there is not time to refer to, tend to show that whites can never permanently and continuously occupy the tropics, especially now that the black races benefit equally with the white by the extinction of infective protozoal diseases. Some have even indulged their fancies in prophesying that this extinction will lead to the ultimate wiping of the blonde white races from off the face of the earth.

Whether this may happen or not, the facts before us, and the more immediate conclusions to be drawn from them, are of concern to us in regard to the colonisation of Northern Australia. Here we have a tropical country which is unique in having no native coloured races sufficiently numerous or willing or able to help in developing the country. Unless science comes to our aid in providing some means by which we can resist tropical influences, above and beyond protozoal diseases, and if we are to believe in history and natural selection, it would appear to be inevitable that if we are to continue to hold this fine country we must either let it remain unproductive, or comparatively so, or we must evolve some scheme by which we can develop it by using coloured labour.

We hear a great deal nowadays about nationalisation of medical services. The matter is one of great moment, in which both the public and the doctors are concerned, and it is desirable that the motives and reasons determining our attitude should not be misunderstood.

The tendency here, as elsewhere, is in the direction of State socialism, and in many matters the State is assuming functions and engaging in operations that have, in the past, been carried on efficiently and well by individual or corporate effort.

In Britain, the National Insurance and Invalidity Bill, introduced by the Chancellor of the Exchequer, is the most gigantic attempt at social legislation of modern times. It is estimated that about 16,000,000 of the population of Great Britain will immediately come under its operations if the scheme comes into force.

"There is no wealth but life," says Ruskin, meaning healthy, efficient human life, and it is not necessary to point out the paramount importance, not only to the wealth, but to the happiness and well-being of a community, of maintaining as many as possible of its units in a condition of health and working efficiency. Every individual incapacitated by sickness or infirmity from carrying on his part in the social economy is a drag on it and a source of poverty.

It is as true now as when Disraeli said: "Public health is the foundation on which rests the happiness of the people and the power of the State . . . . . If the population remains stationary, if it decreases yearly in stature, and in vigour, the nation must perish; and that is why I consider that the first duty of a statesman is the care of public health."

Yet, while we have special Ministers for Mines, Agriculture, Public Works, Defence, Labour and Industry, Education, Lands, and Law Administration, the far more important matter of Public Health is relegated to a sub-department under a Minister who has many other matters to attend to. Has the time, then, not arrived when the Department of Public Health should be under the sole care of a responsible Minister, who should, whenever possible, be a medical man, with a permanent under secretary, who should also be a medical man?

The farmer and agriculturist are supplied with the fullest information as to management of their flocks and herds and crops; but practically the only notions the people themselves get on matters concerning health and disease are those they pick up from the misleading, pernicious literature of quack advertisements.

As State socialism and State philanthropy advance, the scope of their operations tends to widen and to embrace the whole care and healing of the sick. To the principle of the propriety of care and medical treatment of the sick poor by the State we readily assent. As humanitarians, we ungrudgingly give this care now, without any thought of remuneration; but if the State charged itself with this work, and organised it on a proper basis, it would be more satisfactory and acceptable to the poor, as well as to the benefit of the doctor.

We foresee a danger, however, unless the operations of the principle are confined to the really poor, and carefully safeguarded. From our experience of the abuse of public hospitals by well-to-do people, and of the way in which persons, for whom such service was never intended, endeavour to obtain cheap medical attendance through membership of friendly societies, we fear that the evil would be extended by a scheme of State-paid domiciliary attendance, unless the strictest measures were taken to assure that it applied only to the needy and deserving poor, and perhaps to those of the class suitable for inclusion in friendly societies, but precluded from membership by reason of chronic ill-health or infirmity. If the State undertook to provide attendance for these, leaving the friendly societies free to continue their good work, and encourage the spirit of self-reliance with the industrial and poorer middle classes and the remainder to obtain private attendance, as at present, then the scheme would be a desirable one, and would relieve the doctors of a good deal of their gratuitous work.

The difficulty the profession in this State had with friendly societies has been, generally speaking, settled in an amicable manner, commonsense having shown the advantage of having mutually satisfied parties to the agreements, but it was not till the profession took a united stand and refused to be exploited that this was brought about.

If, however, nationalisation of medicine means that the State shall provide medical attendance on all, rich and poor, by salaried State doctors, then we emphatically dissent, and we do so, not only in our own personal interests, but in the interests of medicine and of the public, and of the race.

Medical men do not contend that their private and professional interests should be upheld at the cost of those of the public, nor do they make any *ad misericordiam* appeal. The welfare of the community depends largely on the existence of a strong, competent, independent and sufficiently remunerated medical profession, and, without presumption, we claim that the value of our services in the past entitles our views to consideration in any schemes put forth. And yet we find that both in England and here on big social questions the views of the profession are ignored, their opinions not even asked, though we could materially help in their solution, and prevent the adoption of ill-advised schemes of well-meaning legislators and philanthropists. Indeed, on all social questions the advice of the doctor might be sought with advantage. As a recent writer in the *Australasian Medical Gazette* puts it: "The medical profession occupies in the body social an unique position, and one prepotent of great good to the community at large. In the houses of the great, in the hovels of the poor, amongst the learned and the illiterate, the mentally sound, the morally degenerate and unfit, the doctor daily makes his round. All sides of life he sees, the many minor tragedies, grave crises, little comedies, unfathomable affections, unlooked-for kindliness of heart, and callous selfishness. All sorts and conditions of men it is his daily duty to study for guidance in his administration of the sick. When illness or trouble are present in the house, the outside mask of worldly life is discarded, and the true self stands revealed. The medical man, even beyond the clergyman, sees into the heart of the people, and knows and appreciates their desires. Moreover, he is skilled in weighing facts, and careful in drawing conclusions, he is accurate in summing-up, and broad-minded in the ways of the world. As a member of the most liberal of the professions, he sees what others would fail to grasp, and time and again his advice is sought by a friend, in the affairs of the world, not related to his work."

If we do not get this consideration, especially if we can show that the public will be best served by our being consulted, then we shall have only ourselves to blame. The course is to perfect the organisation in the profession, and of the profession, so that we can voice our views, not by way of unreasonable demand, but by insistence on our rights and privileges, and thus save ourselves from effacement, the science and art of medicine from stagnation, and the public from what we believe would not be to their interest or benefit.

The appointment of salaried State doctors would mean interference with freedom of choice of medical attendants, which is a privilege and an advantage that the public would be very unwise and probably very unwilling to part with. There is no disputing the fact that faith and confidence in his attendant is a very important aid to the patient's recovery. Want of confidence, and the feeling that his freedom of choice had been limited would, on the other hand, have a prejudicial effect.

Moreover, the healthy and powerful stimulus of fraternal rivalry would be removed, or lessened, under a universal system of payment by salary, and the usual State plan of promotion by seniority. An all-round deterioration of the skill of the doctor and of the quality of the treatment would result. This would be to the detriment of the public and of progress in medicine.

Further, the possibility, nay the probability, of a man having to depend for an appointment on his standing with this or that political or municipal party would tend to undermine that independence of thought and action so pre-eminently necessary in a doctor, not only when seeking an appointment but in his work. There would be the temptation to please the employer rather than the patient whenever the interests of the two happened to clash.

If the State is to provide attendance for the poor, and for the poor only, the doctor's remuneration should be by fee for work done, and not by salary, and the patient should be allowed free choice of attendant from amongst those willing to act. I am aware that objections have been raised to this course, but they are easily met. It would take too long to go into the matter now.

The practice of the profession should be left free to follow its natural development, giving the best men the opportunity to advance the science and art of medicine, and to reap the honours and rewards, without which men of capacity and of the right class will seek other avenues for their abilities, and the profession will degenerate.

This is a commercial age, but we are jealous of any radical changes that would abrogate the altruistic or benevolent side of medicine, one of its traditional glories, and reduce it to a purely business basis.

The general practitioner, the consultant, the specialist, all have their sympathies involved, and what Osler calls the "good" debts of the profession amount to a goodly sum at the end of the year. Apart from private benevolent work, there is the large amount done in the public hospitals, by which not only the patients themselves, but the State and the taxpayer benefit enormously.

I doubt if people realise the money equivalent of the work done by doctors in the public hospitals alone. Taking the services rendered to the two chief public hospitals of this city—Prince Alfred and Sydney—only, and calculating the value of the attendance and operations at ordinary fees, it is found that it amounts to no less than £400,000 a year.

If the figures were gone into for the other metropolitan and suburban hospitals the total value of gratuitous medical work would probably be shown to be not far under a million pounds per annum. This does not include the services rendered in provincial hospitals.

The public and the State are, as it were, presented with this sum, to the great saving of the public purse. If this service were not gratuitously given by the doctor the State would have to pay for it. If it were given to salaried servants, it would probably cost much less, but still it would amount to a very considerable sum, especially if salaried officers were appointed to every hospital, city and country, in the same numbers as are found necessary for efficient service; but in proportion as the service would be cheaper, it would be less good.

Under existing conditions the hospital staffs comprise men of the highest skill and standing in the profession, who would neither work as salaried Government officers, nor would they continue to give their services gratuitously if the hospitals were nationalised. We do a good deal of benevolent work for nothing, but we could hardly be expected to be the only people to work for the State without pay. The financial question will, therefore, have to be very seriously considered by any Government proposing to nationalise either the profession or hospitals.

Moreover, hospitals that train medical students would lose the services of the best and most experienced men, and the character of the teaching would deteriorate.

Last Congress gave very careful consideration to the hospital question, and discussed it at length, and if the recommendations then made were adopted a great deal of the overcrowding of public hospitals would cease, revenue would be equal to expenditure, and most of the existing trouble would disappear. The vital principle approved was that no payment whatever should be taken from patients treated at hospitals maintained by the donations of the charitable, combined with Government grant. This would relieve these hospitals of all but the necessitous poor. Others would be provided for by private or intermediate hospitals, the latter being for a class not able to afford ordinary private hospital attendance but able to bear a moderate expenditure.

Though the immediate business of the doctor is to fight disease with the best weapons to his hand, there is an awakening sense of responsibility to the race as well as to the individual. This is shown not only by concern at the effects of the falling birth-rate, but the activities of the Biometricians with their statistical methods; of the Mendelians, attempting to formulate a theory upon the inferences of unit segregation and gametic purity; the plan of the Eugenists to improve, or at least prevent degeneration of, the race by selective breeding—are all indications that medical science is not oblivious to its responsibility.

In the declining birth-rate and race suicide we see evidence of a loosening of the moral fibre of a community, nurtured in self-indulgence, breaking away from the guiding and restraining influences of religion and finding no other monitor.

Leaving the religious, moral, social, economic and patriotic aspect of the question to others, I must confine my remarks to the medical point of view, and even here one is hindered from speaking plainly to an audience such as this by feelings of delicacy and a sense of public decorum.

Amongst medical men there is a growing conviction that many of the nervous complaints of the modern woman arise from repression of her natural instincts and physiological processes, and a suspicion that the measures of restriction adopted may be a cause of female cancer.

What can medical men do to check the evil? We can press the claims of the mother before, during, and after childbirth. We can point out the risks and probable consequences of attempting to defy nature. We can resist the frequent importunities to destroy antenatal life. By care of the new-born child we can lessen infant mortality. We can urge on educational bodies the desirability of plain speaking and instruction on some of the at present tabooed branches of physiology in the schools. We can impress on Governments and our patients the serious consequences of enthetic disease, which in one form results in great infant mortality, or lifelong infirmity, or special susceptibility to other diseases; and in another is the cause of probably four-fifths of the cases of unpremeditated sterility, and, incidentally, is responsible for most cases of blindness, the greater portion of the work of the gynecologist, and productive of untold domestic misery. These effects the average man or woman utterly fails to realise, or else treats lightly.

The best hope of remedying the present drift appears to lie in education, and in elevation of public sentiment. Repressive legislation would probably be of no more effect than were the stringent measures of the Emperor Augustus. Wise enactments might do good, but unless framed with the greatest care and circumspection, and with assistance from the wisest heads in the medical profession, they could easily do more harm than good—indeed, have already done so.

When the figures for the birth-rate are analysed, it is found that the falling-off occurs chiefly amongst the thrifty and provident, the physically and mentally desirable classes, not amongst the thriftless and improvident and degenerate; so that the race is rapidly recruiting itself from below. Statistics give the birth-rate per family of the unfit, or degenerate, at 7·3, compared with 4·5 of a healthy stock. In three generations, 1,000 members of the more desirable classes would be represented by 687 descendants, against 3,600 of the unfit and feeble-minded.

According to latest conceptions, the facts of reproduction and evolution go to prove that life has been carried on by multiplication and perpetuation of the germ cells, or germ plasm, which, with its infinite capacity for variation, has existed since life first appeared upon earth, and which never dies, and, therefore, is in reality immortal.

On this theory of the continuity of the germ plasm, the other cells which form the bulk of the body, the soma cells, or soma plasm, serve as a sort of temporary tenement for the germ cells, hereditary tendencies being implanted in the germ cells only. The offspring is not an individual growth from the parent or two parents, but derived from the fusion of their germ plasm, which was in turn derived from the germ plasm of *their* parents, and so on, back to the first man, As John Morley says: " Every man of us has all the centuries in him."

On this theory it is only qualities that exist in the germ cells that can be reproduced, all other characters being acquired and non-transmissible to offspring.

The theory of heredity that is in accord with all known facts, and which should meet all objections, and be satisfying to biologists, biometricians, and Mendalians alike, is that it is the capacity or potentiality for certain characters or acquirements—what has been called educability—that is germinal, and the direction and extent of the ensuing development depends on the nature and degree of the influences to which the organism is, after conception, subjected, acting on the potentialities of each individual.

Evolutionists believe that man has attained his present position by a long struggle against inimical conditions through the æons of time, the paramount, if not the sole, factor being the gradual process of natural selection, and the elimination of those beings unfitted for their environment. This is the only process of racial development of which we have any knowledge.

The chief adverse condition against which evolution has had to contend, the most stringently selective factor, is disease. We talk glibly of the annihilation of disease, but its possibility is not so very clear. We have no evidence that any disease that has ever existed has been abolished, nor that any new diseases have come into existence within the history of man. Bilharzia has been found in an Egyptian mummy, and a fossil glossina (the conveyor of the protozoon of the dreaded sleeping sickness) has been unearthed in Meiocene deposits. Moreover, if it were possible to abolish some of the diseases we are attempting to destroy, biologists and students of heredity are at variance with humanitarians as to whether it would be to the benefit of the race. For example, while the latter are striving to abolish tuberculosis, others are of opinion that this disease " forms a rough, but on the whole, very serviceable check on the survival and ropagation of the unfit, and that if to-morrow the tubercle bacillus were non-existent, it would be nothing short of a national calamity." (Hunter.)

Barr remarks that this opinion is not likely to command public acceptance, because there is nothing in the present day that people hate so much as the truth when it conflicts with popular sentiment.

Because of its terrible ravages amongst the white races, tuberculosis has been called "the great white plague," but the fact remains that other races, such as American Indians, negroes, Polynesians, Australian aborigines, &c., are not only far more susceptible to it, but show an enormously greater mortality—whole tribes, whole nations having been wiped out of existence by it. We know that tuberculosis has existed among European races for thousands of years, and believe that their relative insusceptibility to the disease is due to their having evolved a relative immunity, those with hereditary susceptibility having been steadily eliminated.

Pathologists tell us that the microbe is everywhere around us, and that in every adult post-mortem the results of bacillary infection can be demonstrated. Yet, though if now amongst whites one in eight dies of tuberculosis, the other races, who have not through exposure to it evolved any immunity, die in the reversed proportion of eight to one, and if they lived under the same conditions as Europeans, as regards climate and overcrowding, their mortality would be even greater. On the other hand the dark races have been evolving a relative immunity to malaria and other parasitic diseases to which whites succumb.

We see the working of the same plan of nature in regard to other zymotic diseases and to narcotic poisons. In countries where plague and cholera and measles are endemic their effects are mild compared with the awful devastation that has accompanied their march to other countries in the epidemic form.

As an agent in natural selection alcohol is believed to be as stringent, if not more stringent, than tuberculosis, weeding out the susceptible and leaving the propagation of the race, in great measure, to those on whom the craving for alcohol has no hold. History teaches us that nations to whom alcohol has been most accessible and who were at one time the most drunken, are now the most sober in the world, as, for example, the inhabitants of southern Europe and the Jews. On the other hand, its ravages amongst communities not prepared by evolution are appalling. Amongst our own race inebriety is most common amongst the poorest classes, who for generations have been the least able to procure it.

We see the same thing in regard to other narcotic poisons. Opium, for instance, has been produced in India for many centuries, and is within easy reach of the people, yet natives never take it to excess. We know its terrible effects upon people to whom it is new, such as the Chinese.

The factor that makes the drunkard is believed to be mainly hereditary, and, along with the tendency to nervous instability so often leading to or associated with alcoholism, to be transmissible to offspring.

Whilst opinions differ as to the harmfulness of alcohol used in moderation, there is no question that taken in excess it is damaging to the individual, as well as productive of much misery, poverty, distress, and crime, and a potent cause and a serious complication of disease. But while this is so, there is no proof of the claim that it causes racial degeneration, but all the evidence is the other way.

Whilst all agree that measures that can be proved to work for the permanent lessening of intemperance should be encouraged, the difficulty comes in deciding as to what sort of measures should be adopted.

Nature eliminates drunkards. Temperance reformers, well-meaning and deserving of all praise though they be for their efforts, but ignoring the lessons of evolution and history, seek to eliminate drink. This seems hardly possible in a civilised community, where anyone can easily manufacture alcohol if he cannot buy it, or he can substitute some still more poisonous narcotic. (Read.) We see already how inebriates, prevented from getting alcohol in a purer form, are taking to drinking methylated spirits.

The sane and proper course would appear to be to segregate the inebriate with the feeble-minded, and prevent him from propagating his kind, rather than to try to eliminate drink by the Sisyphean task of legislating so as to make it difficult for him to get it. The teachings of evolution and history, and our knowledge of human nature, all go to show that the latter course, though it may deceive us by apparent temporary beneficial results, tends in the end not only to perpetuate but to augment the evil by encouraging an increase in the number of persons not protected by evolution and heredity.

If we, then, as medical men are concerned only with the care of the individual—if our laws are made to the same end—if we oppose what Haeckel calls medical selection to natural selection—if, especially in view of the artificial restriction of the birth-rate amongst the most desirable, we go on nursing defectives, and encourage or allow their disproportionate increase, when natural selection works by destroying them,—then, though for a time we may try and deceive ourselves into believing that we have found a better way, it is only a question of time before Nature again takes charge of the process, and the nation goes under to some other whose ways are more in accord with Nature's methods. As Horace says—" You may drive Nature out with a pitchfork, but she will return again."

The popular saying that prevention is better than cure is a dangerous half-truth. Cure means in many cases an after immunity. Prevention, where it acts by increasing the proportion of non-immunes and weaklings, reduces the average vigour of the race, and will add enormously to the future burden of sanitary reformers.

In the present state of public sentiment there are probably not many prepared to subscribe wholly, either to that plan of eugenics, proposed by Plato, and recently revived by Mr. Bernard Shaw, or to the modern form, of which the late Sir Francis Galton and Professor Karl Pearson are the chief protagonists. The primary idea of this doctrine is selection for parenthood, based on the facts of heredity, and it is propounded in two forms:—*Positive eugenics*, meaning the deliberate mating of the most fit, after the manner of the stock-breeder, which would divide humanity into two heterogeneous divisions, and not accord with nature's plan of natural selection; and secondly, *negative eugenics*, which would refuse the right to marry and multiply to the unfit. This latter proposal appears, at first sight, to be reasonable. It might be a step in the right direction if parents or guardians of the contracting parties to marriage, or the parties themselves, were compelled to produce a certificate of freedom from certain diseases before marriage. It is an anomaly that while we inquire into the social or financial position of the parties, we are generally careless in respect to the far more important matter of health. We should not let too much sentimental regard for the liberty of the individual blind us to the responsibility of maintaining the vigour of the race.

The evolutionist of every school, and all students of heredity, agree that the tendency to tuberculosis and alcoholism, mental degeneracy, insanity, feeble-mindedness, idiocy, epilepsy, &c., is hereditary, and that certain other diseases are transmitted from parent to parent, and from parents to children.

The one course which appears to be approved by biology and morality and humanity alike is, while caring for the individual degenerate, or transmitter of disease or infirmity, to prevent such individuals from multiplying. We can agree with the humanitarian who maintains that it is our duty to see that every child born shall be cared for, and with Nietsche to the extent that to abrogate or reverse the principle of natural selection is to court racial death, without going with him so far as to agree that infant mortality is a blessing.

But, though we may agree that the perpetuation of degenerate characters is undesirable, some difficulty appears when we come to decide as to what types should be eliminated. Would our limited knowledge justify our taking the responsibility? Spencer warns us of that practical atheism which, seeing no guide for human affairs but its own limited foresight, endeavours itself to play the god, and decide what will be good for mankind and what bad.

May not the undesirable characters of certain types be correlated with others for the absence of which the world would be the poorer? "Man does not live by bread alone," nor have science, art, music, poetry, literature, philosophy, &c., been the product of only the physically or mentally sound. Many philosophers, poets, scientists, economists, historians, and warriors have been tubercular, alcoholic, or insane or degenerate—John Stuart Mill, Haine, Chopin, R. L. Stevenson, Schumann, Nietsche, Rousseau, Conte, Edgar Allen Poe, to name only a few; while such makers of history as Julius Cæsar, Mahomed, Napoleon Bonaparte, St. Paul (probably), and Alexander the Great were epileptics, and the last was a drunkard, as have also been many men of great intellect and capacity who have made their mark in the world.

It would appear that it would have been far better to have let Nature manage this business in her own way, but as we have ventured to interfere with the balance by restricting the natural increase of the fit, it seems that we are driven to the adoption of some measures to limit the undue multiplication of the unfit, or else we must face logical consequences, which Huxley calls the "scarecrows of fools, but the beacons of wise men."

Here we have entrusted to us a young country of immense possibilities, as yet comparatively free from degenerate stock. Let us try and keep it so, and maintain all that is best and noblest in our people. Though "behind dead Empires the ghosts of dimmer Empires loom," and prophetic vision glimpses a far-off future for mankind, in our brief span we can accomplish but little. Let that little be of the best that is in us, and

Trust that somehow good will be the final good of ill.

We stretch lame hands of faith, and grope
And gather dust, and chaff, and call
To what we feel is Lord of all,
And faintly trust the larger hope.

Hon. G. H. BUTLER: I feel it my duty, first, on behalf of my medical brethren, to assure our worthy President that he may remove from his mind all doubts as to the wisdom of the Congress in electing him President three years ago. (Applause.) We ourselves are fully satisfied with our choice, and we feel that we could not have had a more worthy President on the present occasion. (Hear, hear.) Happily, Dr. Pockley knows too well the troubles and trials of medical men in the present day. With those troubles and trials he has helped to deal thoroughly, both from medical and technical points of view, from a point of view which interests not only ourselves, but also all those who have anything to do with the management of hospitals. In that technical part of his address he has given us much food for thought, of which we will all have to avail ourselves, and which we will be able to read through very carefully (when it is printed) in our studies afterwards. To those interested in hospitals and hospital management, to those attempting to improve the efficiency of hospitals, I commend, for earnest consideration, his remarks in that connection. I am certain all medical men will feel that the poor have to be looked after by those who are able to do it, as Dr. Pockley says, from their own wish to do good, and for that reason alone, and when we once begin to treat the patient by Act of Parliament we will fall in.

Now, ladies and gentlemen, I ask you all to give Dr. Pockley a most hearty vote of thanks for his address this evening. (Great applause.)

PRESIDENT: I have now to call on Sir Philip Sydney Jones to propose a vote of thanks to His Excellency the Governor-General for honouring us by coming here this evening and opening the Congress, and thereby showing his interest and goodwill towards us.

Sir PHILIP SYDNEY JONES: Your Excellency, Mr. President, Ladies, and Gentlemen,—The hour is so late that I feel disinclined to say many words; but we cannot allow His Excellency the Governor-General to leave here without some expression of our thanks for his kindness in coming here and declaring this Congress open. I think the whole community is gratified by the fact that His Excellency has paid this Queen City of the South so early a visit after his arrival in Australia, and we are thankful to him that he has brought with him Her Excellency Lady Denman, who now graces these proceedings with her presence. (Applause.) We trust that His Excellency's administration of the Federal Government will be a very successful one, and that when his term of office is over, he will be able to look back upon it with great satisfaction, and with a conviction that he has endeavoured to live up to the motto of his house: "*Prudentia et constantia.*"

It is as members of the Australasian Medical Congress that we desire to record our thanks to His Excellency. We all know how great an interest our late beloved Sovereign King Edward VII felt in the cure of disease, and in the medical profession generally—an interest which he showed by permitting himself, when Prince of Wales, to become enrolled in our ranks, becoming an honorary fellow of both the Royal Colleges of Physicians and Surgeons in England. We are happy to know, too, that His Majesty's representatives in Australia have not been behind-hand in affording their encouragement to our Congress.

From the very first Congress, held in Adelaide in 1887, up to the present, the Governors and Governors-General have afforded us the honour of their patronage, and in very many instances their presence. His Excellency, by his presence here this evening, and by the words that he has uttered, gives us great encouragement to go on with our work of unravelling the mysteries of disease, perfecting the measures of prevention, and of relieving the suffering. We feel greatly indebted to him for his kindness in coming here.

I beg to move that a most cordial vote of thanks be accorded to His Excellency for coming here and declaring this Congress duly open. (Loud applause.)

LORD DENMAN: I am very much indebted to Sir Philip Sydney Jones for the very kind words said about Lady Denman and myself. I have listened with interest to everything, and I can assure you that it has been a great pleasure to us both to attend this gathering.

(The function then terminated with the National Anthem.)

AUSTRALASIAN MEDICAL CONGRESS.

(NINTH SESSION.)

FINAL MEETING.

HELD AT

THE ROOMS OF THE BRITISH MEDICAL ASSOCIATION,
ELIZABETH-STREET, SYDNEY.

SATURDAY, 23RD SEPTEMBER, 1911.

Dr. F. ANTILL POCKLEY (President of Congress) in the Chair.

The PRESIDENT of Congress: With regard to the reports, I am sorry that we have not been able to get them printed, as we have only been able to get copies of them this morning. The Secretary has some correspondence which he wishes to read before we take the reports from the sections.

CORRESPONDENCE.

Dr. PALMER: I have a letter from the Dental Council of New South Wales, dated 19th September. It reads:—

The President, Medical Congress, Sydney.—Dear Sir,—We look upon this as an opportune time to bring before your learned body one or two matters of great importance to the Dental Profession of Australia with the hope that time may be given by you for a discussion on the points raised by us; we refer:—(1) To the examination of the teeth of the compulsory trained cadets as part of the medical examination. Now, Sir, we contend (and have already protested to the Government) that it is not part of the duty or professional training of a medical man to examine the teeth of these cadets; any more than it is our duty or qualification to examine the eyes of our patients. We are aware that representations have been made by the B.M.A. to the Minister for Defence in reference to the inadequate remuneration paid to the medical examiners under this scheme, and we have also pointed out to the Government that we consider that the members of your profession are already underpaid for medical examinations without taking upon themselves the examination of the teeth; and we now desire to place before your members the injustice still being perpetrated upon a body of men, who claim that the profession of which they are members is at least, if not equal to the Medical Profession, emerging from under a cloud and aspiring to that high pinnacle of learning upon which the Medical Profession to-day stands. The remuneration denied the Dental Profession by these examinations is trivial compared with that which is most dear to us as a body. We refer to official recognition of our profession by the powers that be, which exists in all other civilised parts of the globe. Might we be permitted to point out that the Federal Government is asking and obtaining from the Medical Profession something that, as individual members of unions, they would not permit the most menial members of a trades union to perform, viz., to ask a craftsman of one trade to do the work of a member of another at cut rates or fees. We know that your members have felt the effects of the unethical and disgraceful practice of certain chemists, who, whilst being unqualified, offer consultations and prescribe for the unfortunate and ignorant public. So serious has this matter become in Victoria, that the B.M.A. branch there has entered its protests against this practice to the Pharmaceutical Society of that State, and, whilst we agree with you in this, it is, we consider, only right and just that the same consideration should be shown to others. (2) The next matter we would draw your attention to is the administration of anæsthetics for unregistered and unethical dentists. We are sorry to have to admit that such are to be found in our ranks to-day, but with the co-operation of the Medical Profession, much can be done to eradicate this evil. We would suggest that where a reputable dentist is in practice in any town or village, medical men should refuse to work with the class referred to. We can assure your members that the Dental Profession, comprising this Council, and the Councils of all other States, would not under any consideration, seek the aid of an unethical medical practitioner. We trust, Sir, that you will be so good as to bring these matters before your members and allow a little time for discussion. Thanking you in anticipation, and wishing your Congress every success, We remain, Yours faithfully, R. W. Cleary, Vice-President, W. R. Fitzsimons, Hon. Secretary.

Dr. J. W. **Barrett** (Victoria): I move:—

> "That this proposal be postponed to the Standing Committee of Congress, if formed; if not, to the local Branch of the B.M.A."

The motion was agreed to.

RECOMMENDATIONS FROM SECTION OF OPHTHALMOLOGY.

Dr. J. W. **Barrett** (Melbourne): I have to bring up the report of the Section of Ophthalmology for your endorsement:—

> "That this Congress sympathises with the movement for the better education of those who wish to become spectacle makers, and is of opinion that a proper course of technical instruction should be provided for those entering the trade. It is suggested that such instruction can be provided by arrangement with Technical Colleges, and that members of the Congress will assist in effecting such arrangements in their respective States.
>
> "That the Congress is of opinion that in the interests of the public no legislation should be introduced which suggests that opticians should be legally authorised to test vision. The relationship of the optician and the oculist should be that of the pharmaceutical chemist to the physician.
>
> "That the term 'oculist' should not be assumed by any person who is not a legally qualified medical practitioner."

The motion was carried.

HOSPITALS COMMITTEE.

Dr. J. W. **Barrett**: This is a very important matter, Mr. President and Gentlemen, and comes from the Hospitals Committee of the Congress. The Committee is unanimously of opinion that the nationalisation of the profession is within sight, if not clearly imminent, and they think that by one strong step, and one strong step only, that can be really obviated; that is embodied in the following resolution:—

> "That a copy of these resolutions be forwarded to the State and Federal Governments of Australia and to that of the Dominion of New Zealand.
>
> "That this Congress is of opinion that active and immediate steps should be taken to establish intermediate hospitals, with the object of relieving the public hospitals of those patients who are able to make some contribution.

> "In the opinion of Congress it is necessary that such intermediate hospitals should be absolutely controlled by the medical profession.
>
> "That these resolutions be forwarded to the Councils of the various branches of the British Medical Association in Australia and New Zealand, and that the Councils be requested to give the matter their immediate and earnest attention."

The answer made by Mr. Flowers to us was perfectly clear that, you must provide for the person who can pay something before limiting a hospital to those who can pay nothing. This represents the unanimous opinion of the Hospitals Committee.

Professor ALLEN: Not that we recognise that such a state of things is really going to come; merely that the movement is on foot.

Dr. WORRALL: The proposed nationalisation of hospitals on the lines laid down by Mr. Flowers would be nothing less than a calamity to this profession, and, therefore, prejudicial to the public interest. The suggested intermediate hospitals must be controlled by the British Medical Association, or evil will result worse than it is sought to cure. With this safeguard the establishment of such institutions is most desirable, as their absence is the one weak spot in the case for the profession in defending itself against the schemes for nationalised hospitals admitting rich as well as poor.

The motion, on being put to the meeting, was carried unanimously.

RECOMMENDATIONS OF PUBLIC HEALTH COMMITTEE.

Professor BERRY: I move:—

> "That it is the opinion of this Meeting that the notification of cases of infantile paralysis should be made compulsory, and that a recommendation be sent to General Congress that they should notify the passage of this resolution to the various State Governments."

This decision was come to at a conjoint meeting of the section of neurology, pathology, public health, and diseases of children.

Professor ALLEN: I second that.

Carried unanimously.

Professor WILSON: On behalf of Professor Berry I have the honour to move the following resolution which has already been passed by the Public Health Section, and which has received the full consideration:—

> "That this Medical Congress endorses the proposals of the Anthropometric Committee of the Australasian Association for the Advancement of Science, that an anthropometric survey of Australasian children be undertaken."

At the last meeting of the Australasian Association for the Advancement of Science, in connection with their section of Anthropology, a committee was appointed to consider the problem of an anthropometric survey of children, especially in regard to the children of Australasia. That Committee had a meeting during the sitting of this Congress, and

Professor Berry was deputed to move at this meeting that the Congress endorses the proposal. It is a matter of some public importance, and in approaching the Federal Government or any other Government, it is a step which will have to be taken, and the approbation of this Congress would be of very great value.

The motion was carried.

Dr. W. G. Armstrong: Mr. President, Ladies and Gentlemen,—In the absence of the President of the Section of Public Health, I have been asked to move the following resolutions as Secretary of the Section. There are a number of them. The first is:—

> "That in the opinion of this Congress a time will come when the compulsory notification of syphilis and gonorrhœa will be necessary, and the earnest attention of the Health Department of the Australian States should be drawn to the matter, with the object of introducing such notification when the time is ripe."

Dr. Lendon: An investigation scheme is under way by the Tropical Institute.

Dr. Armstrong: That is a motion carried by the Section of Public Health for the consideration of the Congress.

Professor Allen: I move an addition of the words:—

> "And that the Congress express its sense of the importance of having such work done."

I should like to have a copy of the resolutions, because I will be approaching the Government in connection with the Tropical Institute, and I could use them then.

President of Congress: There are five resolutions. I will now put to the meeting the first resolution in the amended manner suggested by Professor Allen:—

> "That in the opinion of this Congress a time will come when the compulsory notification of syphilis and gonorrhœa will be necessary, and the earnest attention of the Health Departments of the Australian States should be drawn to the matter, with the object of introducing such notification when the time is ripe, and that the Congress express its sense of importance of having such work done."

Carried, with one dissentient (Dr. Guy Griffiths).

Dr. W. G. Armstrong: The next resolution from the Section of Public Health is:—

> "That the Executive of the Australian Medical Congress advise each State Government of the necessity for obtaining the exact number of the feeble-minded in each State."

Dr. Lendon: Where are we going to draw the line, Mr. President?

Professor Allen: Inquiry should be made as to the number of feeble-minded in each State who require special instruction and education, and all necessary steps should be taken to see that such special education is given. I move:—

> "That the Congress advise the State Governments to inquire as to the number of feeble-minded children needing special education, and to take steps for the provision of such education."

Carried as amended.

Dr. W. G. ARMSTRONG: I beg to move the adoption of the following further recommendations:—

> "That it is desirable, in the opinion of this Section, that certificates of death should be given under cover to the responsible person in the household of the deceased to be delivered sealed to the Registrar of Deaths, and that a letter be addressed to the Prime Minister of the Commonwealth, asking that the Premiers of the States be urged to bring this about by such steps as are necessary."

The motion was carried.

> "That with relation to the resolution dealing with the collection of available Australian Medical data and statistics already carried by Congress, a central committee be formed consisting of Dr. Ashburton Thompson, Mr. Knibbs, Dr. W. G. Armstrong, and Dr. Frederick Watson, for the purposes of the resolution, and that in each of the other States the Chief Medical Officer of the Government should be asked to nominate a local committee."

The motion was carried.

> "That the Congress is alive to the importance of the urgent need for the further investigation of the relationship of mosquitoes to disease in Australia, and for the classification of mosquitoes round the coast."

Carried.

> "That it be a recommendation to Congress from the Section of Public Health, that elementary hygiene be made a compulsory subject of instruction in State Schools for all children over the age of 12, and that the feeding and rearing of children be also made a compulsory subject for all girls over the age of 13."

Dr. LENDON: Can we not refer that to a committee to report at the next Congress?

Dr. J. W. BARRETT: The matter has already been considered in Victoria by the Education Department.

Dr. HONE (South Australia): It is a very big subject; but a recommendation from this Congress cannot do any harm.

Dr. J. W. BARRETT: The fixation of the age is the difficulty; if it were more general there could be no possible objection.

Dr. W. G. ARMSTRONG: I might cut out all reference to age and make it "all children."

PRESIDENT OF CONGRESS: "All children" is understood.

Dr. W. G. ARMSTRONG: "That elementary hygiene be made a compulsory subject of instruction in State schools, and that the feeding and rearing of children be also made a compulsory subject for the older girls." How would that do?

Dr. ARTHUR: I should like it to be put to the vote. I think it is a most important part of the resolution, and this matter, so far as State schools in New South Wales are concerned, has been in operation at least six or seven years. A medical officer was appointed by the

Government here to carry out the terms of that paragraph in regard to the instruction of the older girls in schools in the care of infants. I think, if consideration is given to the matter, that the meeting will see the wisdom of it.

Dr. HURST: It is a long resolution. The first part includes everything we want, and will cover all the last speaker has referred to.

Dr. J. W. BARRETT: Would it not meet the difficulty if, after the word "hygiene," you put "including the feeding and rearing of children."

A MEMBER OF CONGRESS: That is not worth while.

Professor ALLEN: Do not make an opening for the learning of boys in regard to the latter part, or you will have them taught to sew.

Dr. ARTHUR: I move that the resolution, as originally read, be adopted.

A MEMBER OF CONGRESS: What, with the ages in?

Dr. ARTHUR: With the insertion of the last paragraph which it is suggested should be eliminated.

PRESIDENT OF CONGRESS: I do not quite follow. I do not think anyone suggested the elimination of the last paragraph.

Dr. W. G. ARMSTRONG: In the original motion the paragraph reads:—"That the feeding and rearing of children be made a compulsory subject for all girls over 13." It has been suggested that it should be altered to read—"for all the older girls."

PRESIDENT OF CONGRESS: Do I understand that Dr. Arthur now moves that that be an amendment to the resolution?

A MEMBER OF CONGRESS: Why not put the resolution in two sections?

PRESIDENT OF CONGRESS: It is practically the same thing.

Dr. HURST: It is quite true that the greater includes the less. I think it is better to omit the ages.

Professor ALLEN: Is there a unanimous feeling? Let it go. Let it all be done in full.

PRESIDENT OF CONGRESS: I will now put that to the meeting—that the resolution, as originally drafted, be adopted.

Carried.

PRESIDENT OF CONGRESS: That was the original resolution. We will now put it back, with this alteration to the latter part:—"And that the feeding and rearing of children be also made a compulsory subject for the older girls."

The motion was carried, with four dissentients.

Dr. W. G. ARMSTRONG: The next point is:—

> "That this Section recommends to Congress to advise each State Government to introduce the provisions of the Prisoners' Detention Act, 1908, N.S.W., and the provision of the Police Offences Act, 1908, N.S.W., relating to venereal diseases, with the substitution of 'any person' for 'common prostitute' in the latter."

President of Congress: I do not know whether we all know what that means. You must give some brief explanation of it. The clauses of those acts referred to in this resolution relate to the detention of any convicted prisoner who is found to be suffering from any venereal disease until, by the results of medical treatment, the prisoner is cured. He or she may be detained under treatment until a cure is effected.

Dr. Clarence Read: At the present time it does not go far enough. If persons are convicted they are given the option of a fine, and if they pay the fine, although they may have a venereal disease, they cannot be detained. For instance, we will say a man or woman is sentenced to seven days' imprisonment with the option of a fine of 10s. The fine is not paid. They go to prison for seven days, and, at the end of the seven days, they cannot be detained. I think that is so. We want the resolution to go further. It does not go far enough.

President of Congress: This is a matter which is certainly contentious, and, without expressing an opinion one way or the other myself, there is undoubtedly a controversial element in it. This matter was discussed pretty fully in the Section of Public Health; but the other men who did not hear it discussed might not be prepared to vote one way or the other on matters which they do not fully seize.

Dr. J. W. Barrett: Do I understand that this practice is actually in force in New South Wales at the present time?

President of Congress: It is in force; but, in the view of many, and for the reasons stated by Dr. Read, it does not go far enough.

Professor Allen: That was thoroughly threshed out, and we had evidence before us, quite convincing, that good results were being obtained by the administration of such Acts in the States that have them. I urge the Congress to accept the decision that has been come to.

Dr. J. W. Barrett: In Victoria, the Principal Medical Officer has drawn attention to this mode of dealing with venereal diseases, and, without indicating his approval of this proposal, he has indicated the necessity of something of the kind.

Dr. Arthur: Dr. Armstrong might explain to us why "any person" should be substituted for "common prostitute."

Dr. Armstrong: The clause in the Police Offences Amendment Act, I believe, provides that any common prostitute may be detained; whereas, by this resolution, it is proposed to substitute for the words "common prostitute" "any person," in order that any person of either sex might be detained. That is perfectly clear, I think.

Dr. R. H. Todd: I think it is very difficult for us to understand the meaning that Dr. Armstrong is trying to make clear to us, unless we have something definite before us. I do not see, myself, how in these circumstances they are going to detain anyone except someone who is in a position to be detained. Unless a common prostitute comes before the Court in some way or other so as to be in a position to be detained, I cannot see how they can pick out a person in the street and say: "You are a common prostitute, come along with me. I am going to hold you until the doctor has cured you." It is absolutely impracticable.

I do not understand it, and with regard to Dr. Read's suggestion, he points out that so far, the Act, as it is at present, is not effective, because, when a person is convicted of an offence and fined instead of being sent to prison, he is not detained. It is practically an impossibility for the police or anyone else to detain a person or anyone at all who has not been sentenced to imprisonment, or who is merely fined. If the person pays the fine he or she is free to go away. I see considerable difficulties in the matter myself; but, I must confess that, in the way it has been put to the meeting, I do not quite understand what the effect of it will be.

Dr. PURDY (New Zealand): This resolution was unanimously passed by the British Medical Association in New Zealand at their last Congress. I cannot, for the life of me, understand the difficulty there is about the resolution. It is so simple. At the present, of course, only a prisoner who is a "common prostitute" could be detained; but, in the terms of the resolution, "all prisoners" could be detained who are suffering from venereal disease. That must be perfectly clear to everybody, or ought to be perfectly clear to everybody.

PRESIDENT OF CONGRESS: Would it do to substitute the word "prisoner" for "person"?

Dr. PURDY (New Zealand): It is understood that a man cannot be detained unless he is a prisoner; it means on conviction, Sir.

A MEMBER OF CONGRESS: If he is fined?

Dr. PURDY: Dr. Read is perfectly right, Sir. It is perfectly clear that there are two ways of dealing with a prisoner; a prisoner can be fined 10s. and the costs of the court, with an option of seven days' imprisonment. That is put in, because, if there is no option, and the prisoner does pay the fine, they cannot do anything to him. Therefore, they always fine a prisoner with the option of imprisonment. If the prisoner does not pay the fine, and elects to go to prison, and he is suffering from a venereal disease, he cannot be detained, because he has been imprisoned. (Cries of "No.") It is correct.

A MEMBER OF CONGRESS: That is not correct.

Dr. PURDY: I tell you it is correct. It is perfectly clear. Everybody knows the difference between a man who is sentenced and fined, and a man who is imprisoned without the option of a fine. If they do not know it, they ought to know it. They ought to take it from me that it is so.

Dr. FOREMAN: I have gone into this subject a good deal. The position is this: Any person who is convicted with the option of a fine, although suffering from a venereal disease, can go out at the expiration of his time, if he elects to go to prison, and the prison authorities have no power at all to prevent it. Anyone, male or female, who is convicted, without the option, can be detained until the surgeon passes them as incapable of spreading infection. The words "undergoing sentence of imprisonment" cover those who really can be inspected or examined. It does not appear to me that there is any power to examine people who are sentenced with the option of a fine, and who elect to pay their fine. I do not see how they can be netted.

Professor Allen: Would it meet the wishes of members if it were put in a more general form? Would it not be better to say that the various State Governments be asked to provide, by legislation, that persons suffering from venereal disease may be detained for treatment?

President of Congress: Undoubtedly.

Professor Allen: They would have to think what they are prepared to do. They would look into all the authorities and practices, and in that way some satisfactory result would be arrived at. We only want to draw attention to it.

A Member of Congress: Would it not be better to make it read:—" The State Governments 'shall' provide," and so on.

A Member of Congress: We cannot say that.

Dr. Perkins: I think you might make an alteration still further, and say, instead of " prisoners convicted," " any prisoner suffering from a venereal disease," because you get the prisoner who is convicted and who pays his fine, under the other suggestion, going scot free, although he may be suffering from a venereal disease. He goes scot free and spreads the disease all over the place. That is what we want to prevent. I think if you would carry out my suggestion it would meet the case. Much more would be accomplished.

President of Congress: The other night we had a discussion as to the meaning of " convicted person," and even with the assistance of Dr. Todd, we could not find it out.

Professor Allen: I think, if we discuss these questions here, we will go on indefinitely. Let us put it to the Government—" Would you read the whole of these resolutions bearing on the subject?"

Dr. W. G. Armstrong: " That in the opinion of this Congress, a time will come when the compulsory notification of syphilis and gonorrhœa will be necessary, and the earnest attention of the Health Departments of the Australian States should be drawn to the matter, with the object of introducing such notification when the time is ripe." That has been already adopted, with the following amendment by Professor Allen—" And that the Congress expresses its sense of the importance of having such work done." (2) " That it should be a legal offence for any person who is not a legally qualified medical practitioner to treat a case of venereal disease." (3) " That each State Government be invited to provide increased facilities for the treatment of cases of venereal disease." (4) " That general hospitals and dispensaries, rather than special or lock hospitals, should provide the necessary accommodation of venereal cases." (5) " That it be a legal offence for any person cognisant of the fact that he or she is suffering from a venereal disease to communicate such disease." And there is the resolution with regard to the Prisoners' Detention Act, which I have already read.

President of Congress: I understood those are all to be carried as one.

Professor Allen: They are all one resolution, really.

President of Congress: It would be undesirable to have one without the other. Do you wish these resolutions to be approved?

The motion, on being put to the meeting, was carried unanimously.

RECOMMENDATIONS FROM THE SECTION OF PATHOLOGY, BACTERIOLOGY, AND TROPICAL MEDICINE.

Dr. LOVE: Mr. President, Ladies, and Gentlemen,—It has been unanimously resolved:—

> "That it be a recommendation to Congress that representations be made to the Federal Government with a view to securing for the Australian Institute of Tropical Medicine such increases of funds and staff as will enable an organised inquiry to be undertaken without delay into the various matters likely to affect the permanent establishment of a working white race in tropical Australia.
>
> (2) "That it be a recommendation to Congress that the permanent settlement of a working white race in tropical Australia be adopted as the principal subject for discussion at the Brisbane meeting, when held."

I have further to move:—

> "That Professor Allen be requested to personally present the Minister with these recommendations."

There is one further recommendation:—

> "That this section strongly recommends to the Committee of the Australian Institute of Tropical Medicine that a comprehensive study of the subject of granuloma pudendi be at once undertaken."

Professor ANDERSON STUART: I beg to second that. I should like to inform the Congress that yesterday, at a Committee meeting of the Australian Institute of Tropical Medicine, we decided to ask the Federal Government to increase the grant to £4,000 per annum, and as the Under Secretary for External Affairs is a member of the Committee, and he was present, we have every reason to believe that the Federal Government will grant our request. We have also asked for the appointment of two assistants under the Director, Dr. Breinl. This is the most important of many questions which I hope will be settled at the Brisbane Congress, and I recommend most strongly to the Congress, that it shall give every facility and every support to the motion, so as to make the Brisbane meeting a great meeting for the settlement of a great and national question. There is no question so pressing as this, and it is nonsense to say it is political; it is not political; it is in the first instance medical, and this Congress must settle medical questions first.

In regard to the mosquitoes, we appointed an entomologist a few months ago, and that entomologist is at the institute at this moment, doing nothing else but mosquitoes.

PRESIDENT OF CONGRESS: If I might make just one more remark from the Chair: Professor Anderson Stuart says the matter is very pressing. I hope that this relegation to six years hence will not mean, because it is coming on then as the principal subject for discussion, that we have no need to do very much before then. Everything that can be done in the interval should be done.

Dr. ARTHUR: Three years ago, at the Melbourne Congress, a resolution was carried and was taken to the Prime Minister, asking that a Commission be appointed to investigate the question of the possible colonisation of tropical Australia by the white races, and although the Prime Minister then promised that some attention would be given to the matter, nothing, so far as I have been able to learn, has been done. I would suggest that, say, Dr. Barrett, who has taken a great deal of interest in this subject, might be asked to convey this resolution which has just been moved and seconded, to the present Prime Minister, and to ask him if his Government will take some cognisance of the resolution passed three years ago.

Professor ANDERSON STUART: If we are going to tackle the subject through the Tropical Institute at Townsville, we had better get that done. Let us get one central organisation going properly, which will be doing this work and nothing else. Let us get that Tropical Institute going on a proper effective basis with sufficient staff and sufficient laboratories, and when that has been done, the work will be developing every day, every month, every year, steadily, until the results are obtained. (Hear, hear.)

Dr. R. H. TODD: At the time Dr. Arthur speaks of, there was no such thing in existence as the Australian Institute for Tropical Medicine, and now that has come into existence, it is a more powerful and a better body to deal with this work than a commission could possibly be.

Dr. J. W. BARRETT: That is not quite correct. The Tropical Diseases Institute is the proper body to investigate the medical aspect; but the Government has not formulated a policy for the settlement of the Northern Territory, which is very largely a medical matter, and it is a question whether the profession, as a whole, in addition to the Tropical Institute, will not have to deal with it.

PRESIDENT OF CONGRESS: Dr. Love in his resolution recommended that Professor Allen should personally carry that to the Minister.

Dr. GUY GRIFFITHS: I should like to move, as an amendment, that all references to a White Australia policy be eliminated from the resolution. This Congress should concern itself with medical matters, not political. This White Australia policy is a selfish, inhuman, and unchristian policy, a policy with which this Congress should not be mixed up in any way.

Professor ALLEN: So far as I am concerned, there will be no reference made to a White Australia policy or anything of the kind; but there will be the strongest effort I can possibly put forward towards the achievement of an exhaustive inquiry as to the possibility of maintaining a settled, working, white population. (Hear, hear.)

Dr. GUY GRIFITHS: I think in the resolution that was moved, the words "White Australia" occur.

PRESIDENT OF THE CONGRESS: No. "Working white race in tropical Australia."

A MEMBER OF CONGRESS: "White Australia" will not be put in.

The original motion, on being put to the meeting, was carried.

RECOMMENDATIONS FROM SECTION OF NAVAL AND MILITARY MEDICINE AND SURGERY.

Dr. PALMER (Honorary General Secretary of Congress): I have received the following letter from Dr. Newmarch, dated 20th September :—

> In accordance with the motion proposed by me, which was seconded and passed at the Business Meeting of Congress held on 18th September, 1911 :—I have the honour to report that the motion was read to the members assembled at the meeting of the Section of Naval and Military Medicine and Surgery held on 19th September, 1911, by me as Honorary Secretary. Further that the letter referred to in the above-mentioned motion, together with the suggestions appended, were also read. In the discussion which followed the opinion was very freely expressed that the scheme, as outlined in the letter and suggestions above named, was absolutely disapproved. The following members took part in the discussion :—Surgeon-General Williams, Colonel Fetherston, Colonel Mullins, Colonel Eames, Major Kirkland, Major Newmarch, and Dr. Prevost. The following resolutions were submitted to the meeting and carried :—(1) Proposed by Colonel Fetherston, seconded by Major Kirkland,—" That the scheme as outlined is not acceptable to the Congress, as it considers the terms of service and pay are quite inadequate to attract any candidate." (2) Proposed by Major Newmarch, seconded by Colonel Fetherston,—" That this Section of Congress is of opinion that to formulate an efficient scheme for the establishment of a Commonwealth Naval Medical Service, that a Committee should be formed to discuss the subject, if possible, with the Naval Board. Surgeon-General Williams and Major Newmarch were nominated to act on the Committee if arranged as above."

I move that those resolutions be adopted.

Dr. CLARENCE READ: I second that.

Carried.

Surgeon-General WILLIAMS: Mr. President and Gentlemen,—The result of the meeting of the sub-committee appointed to inquire into the payment, by the Commonwealth Government, of area medical officers, came to these conclusions which I have now to submit :—

1. That the present arrangements of payment as recently approved by the Government be accepted, and that a fair trial be given to them during the present financial year ; at the end of that time a further report can be submitted to or by the associations.

2. That a special rate of pay be allowed for examination of cadets by civilian medical men (an allowance of 10 per cent. to be done in this way is made).

3. They also report that with the rearrangements of areas now in progress, the numbers to be examined in each area will be more equal.

Those are the three resolutions which the sub-committee, after mature deliberation, were unanimous in passing.

On being put to the meeting, the resolutions were carried unanimously.

STANDING COMMITTEE OF CONGRESS.

REPORT of the Committee appointed by the Congress on the 18th September, 1911, to consider and report upon the motion moved on that day by Dr. J. Adam Dick, providing for the formation of a Standing Committee of the Congress.

The Committee reports that it has considered the motion moved by Dr. J. Adam Dick on the 18th September, 1911, providing for the formation of a Standing Committee of the Congress, and recommends that the Congress resolves as follows:—

" That a Standing Committee of the Australasian Medical Congress, to be called " The Commission of Congress," be and is hereby formed with the following constitution, viz.:—

1. *Membership.*—Such Commission shall consist of the President, the Honorary General Secretary, the Honorary Treasurer, two members appointed by the Council of each Branch in Australasia of the British Medical Association, provided that the President shall cause each such Council to be notified of its right to make such appointments, and that any proceedings of the Commission taking place after thirty days from the date of such notification shall be valid, although all or any of such appointments shall not have been made.

2. *Duties.*—It shall be the duty of such Commission—(*a*) to carry out, so far as in its power lies, such instructions as the Congress shall give to it Session by Session; (*b*) to note the course of medico-political events of importance in Australasia, and, where, in its opinion, intervention by it, as representative of the medical profession in Australasia, is urgently called for, to take such action as it may deem appropriate to safeguard the interests of the profession; (*c*) to be at the service, as an advisory body, in medico-political matters, of the Government of the Commonwealth of Australia and the Dominion of New Zealand; (*d*) pending the adoption, by the Congress, of rules for the conduct of the proceedings of the Commission, to make such rules as it may consider expedient for this purpose. Provided that until the making of such rules, and in any matter not provided for in such rules when made, procedure shall be determined by the President. Provided also that at any meeting of the Commission four shall be a quorum; (*e*) to meet from time to time at such places and times as the Commission, or failing the Commission the President, may fix; (*f*) to offer from time to time such reports of its proceedings as the President may approve, to the *Australian Medical Gazette*, the *Australian Medical Journal*, and such other medical newspapers as the Commission may select, for publication therein; (*g*) in the event of any officer appointed by the Congress ceasing to be able to act while the Congress is not in Session, to fill the vacancy, thereby occasioned, on the recommendation of the Executive Committee of the Congress, or, if there be no such Executive Committee, on the recommendation of the Branch of the British Medical Association in the Dominion, or State, in which the next Session of the Congress is to be held; (*h*) to report to the Congress at each Session thereof in regard to its proceedings since the previous Session.

3. *Powers.*—Such Commission shall have power—(*a*) pending the adoption by the Congress of rules for the conduct of the proceedings of the Commission, to alter, amend, or rescind any rule made by it for such purpose; (*b*) to delegate to committees, consisting of one or more of its members, such particular duties and powers as it may specify in any resolution appointing any such Committee, and any such Committee shall have power, with the express approval of the Commission or the President, to co-operate with any member or members of the profession specially qualified to assist it—provided that no such Committee shall continue for more than three months unless a longer period be fixed by the Commission; (*c*) to form sub-committees of its members with such powers and duties as may be deemed by it to be expedient.

4. *Expenses of the Commission of Congress.*—The necessary expenses, other than the travelling and personal expenses of the members, shall be a charge on the moneys in the hands of the Honorary Treasurer of the Congress, who is hereby authorised to pay the same."

W. T. HAYWARD,
Chairman.

Sydney, 22nd September, 1911.

President of Congress: Dr. Hayward presents a report and wishes Dr. Dick to move the adoption of it. You have it in your hands.

Dr. J. Adam Dick: To save time, and to make the resolutions clear, they have all been printed, and are in the hands of every member present. I beg to move that the report of the Committee of Congress appointed on Monday morning, be adopted.

Dr. Hayward: I second that.

Professor Allen: I should like to make this remark upon it: I hope that a Standing Committee, such as we have here, will take it as one of its powers to enter into communication as much as it would like either with branches of the British Medical Association or with any organisation formed outside of those branches. so that we may get the greatest unity of action of the profession throughout Australasia. (Hear, hear.)

Dr. Kenny (Melbourne): I should like further to add, in extension of the idea to which Professor Allen has just given expression, that in paragraph 1, with regard to membership, some words——

Professor Allen: Do not express things too much. If there is an understanding that the Committee has powers, it will all work out.

Dr. Kenny: It is not a question of powers; it is a question of representation. You see this resolution will confine the representation on the Standing Committee of the Congress to certain executive officers of the Congress and to "two members appointed by the Council of each branch of the British Medical Association." There are several other associations in Australasia and New Zealand, and I ask that the same representation shall be given to those associations.

Professor Allen: I suggest that Dr. Kenny shall move that this committee have power to confer with other associations.

Dr. R. H. Todd: I would have no objection to the clause in that way if I understood what it meant.

A Member of Congress: It is power to add to their membership; that is all it means.

Dr. R. H. Todd: The power Dr. Kenny asks for is practically given in (3) (*b*) "To delegate to committees, consisting of one or more of its members, such particular duties and powers as it may specify in any resolution appointing any such committee, and any such committee shall have power, with the express approval of the Commission of the President, to co-operate with any member or members of the profession specially qualified to assist it—provided that no such committee shall continue for more than three months unless a longer period be fixed by the Commission." Once you get that power you can call in anybody who would be useful to you for any purpose whatever. Under that subsection I say that you have that power already, and I think it would be far simpler if it were left as it is, rather than provide for any special representation for any body calling itself a medical association. Such a thing would be too indefinite altogether. The British Medical Association is the only one that is properly organised. There are several local associations, and they have an existence, perhaps, very useful in their way; but they are not in any way representative of the profession as a whole, and I think, if we gave such an extended representation of the profession as that asked for by Dr. Kenny, we would make this Committee so cumbrous and so expensive that we would never be able to do anything at all.

Professor Allen: I am sure many of us agree thoroughly that there should be no explicit representation—in fact, no representation of any other body whatever; but I do think it would be wise to give this Committee the power to actually have, in its own self, such other persons as it might think flt. It will be entirely composed of representatives of the British Medical Association, and the highest officers of the Congress, and if they have power to bring in such other persons as they think fit, I think then you will have a better working body. (Hear, hear.)

Dr. Kenny: That raises the whole difficulty, in my mind. In one sense that is practically putting in the thin end of the wedge for the insistence that membership of Congress shall be membership of the British Medical Association. (Cries of "No, no.")

Professor Allen: Might I speak to that? To those who have thought this matter out carefully, it has been clearly recognised that membership of the Congress should not be absolutely limited to membership of the British Medical Association; but, if there are persons in any State who are distinctly distasteful and impossible people, they will be excluded. If there are people who have not joined the British Medical Association, but against whom there is no great block, the mere fact that they are not members should not debar them from admittance to Congress; they will be permitted to join the Congress, and we hope that the Congress will be a feeding ground to the British Medical. (Applause.)

President of Congress: This clause does not necessarily mean that the membership of the Council shall be confined to members of the British Medical Association. It says: "Two members appointed by the Council of each branch in Australasia of the British Medical Association." They might possibly or conceivably appoint somebody who is not a member of the British Medical Association.

A Member of Congress: They might. (Laughter.)

President of Congress: It is a most useful thing to have power to add to your number. The Committee would not be likely to swamp itself. There might be some very useful person whom they would like to have the power of appointing, and unless they have that power they would not be able to avail of his services.

Dr. R. H. Todd: They have the power.

Dr. Kenny: I should like to say (and I think those who know me well will appreciate that I have not the faintest scintilla of hostility against anyone belonging to the British Medical Association—indeed, I have spent the years of my best work for the British Medical Association) that I want to see the British Medical Association the largest organisation in all the Australasian States. (Hear, hear.)

A Member of Congress: It is.

Dr. Kenny: But I want to see the British Medical Association the largest organisation on proper lines. I foresee difficulties; I foresee troubles in that connection which are not a mere passing thought of the moment while I am speaking in this room. I gave expression to some of those troubles at the Melbourne Congress. This Congress represents the whole of the profession that is clean-handed, and each member worthy of the name in the total area of Australia and the Dominion of New Zealand should have to guard against anything at all that would be in any way restrictive. First of all, as regards membership. On that point it is contended that we do not want to restrict interest in the Congress. I maintain that, in legislating for future arrangements, you have the right —not only the right, but it is a duty—to take such steps as will bring about a proper, eligible interest. There do exist many other medical associations outside the British Medical. (Cries of "Name them!") And in some States they have a greater relative proportion to the British Medical than they have in this State. My own idea with regard to other States is, the sooner the British Medical Association occupies the prominent position that it does in this State the better, and that can only be brought about by good organisation. When that time arrives I will say that the British Medical Association should be the Congress; but not until then. In order to keep up the widest possible area of interest with this Congress, I say we should take in representation from all the existing medical associations. (Cries of "No, no," and, "There are hundreds of them.") I am merely stating my own opinion. And those who endorse my opinion will endorse it by their votes presently. I am quite prepared for "noes"; there are "noes" on the Committee; and there are instances of "noes" on the Committee who went in "yes." I know that the matter has been discussed very seriously. As one who, in common with you all, feels deeply interested in the future success of the Congress (and it has achieved a great deal of success in the past), I would be lacking in a sense of duty if I did not give vent to my feelings on this matter, and give vent also to the feelings of those who think the same as I do. (Hear, hear.)

PRESIDENT OF CONGRESS: I think we should have the names of those other associations that you mentioned, Dr. Kenny. Three, four, or five men might form an association, and you surely would not give them representation? Kindly let us know the names of the associations you refer to.

DR. KENNY: I am not going to give a list at the present moment. There is a medical association in Melbourne——

A MEMBER OF CONGRESS: That is social.

DR. LENDON: And the bulk of their members are members of the British Medical Association.

ANOTHER MEMBER: We have several associations in this State. (Cries of "social.")

DR. KENNY: My experience is that the majority of these associations are merely social.

PRESIDENT OF CONGRESS: Do I understand you to move an amendment, Dr. Kenny? (Cries of "No.")

DR. KENNY: I should like to move as an amendment that after the word "British Medical Association" in the first clause, under the heading "Membership," an addition of the words "or any other medical association." (Cries of "No.")

President of Congress: Dr. Kenny moves as an amendment that after the words "British Medical Association" the words "or any other association," so that it will read:—

> "Two members appointed by the Council of each branch in Australasia of the British Medical Association, or any other medical association."

On being put to the meeting the amendment was lost.

Professor Allen: I propose the insertion of the words "with power to add to their number," or something like that.

A Member of Congress: Will you limit the number?

President of Congress: I do not think they would swamp themselves.

Dr. Hone: We are to seek it from the side of the Congress and the side of the British Medical Association. We have a Committee appointed on both sides; we have this Standing Committee, and we have in view, I think I may say, a Federal Committee of the branches of the British Medical Association. The more we can get those two committees into line the more it will gradually tend to bring about just that consummation that Professor Allen and Dr. Kenny have expressed this morning. I believe that the members of the British Medical Association could, if they wished, push the federalisation of the branches of the British Medical Association so far that this Congress would be a Congress really representative of the British Medical Association. But they do not want it, because they want to safeguard the interests of those gentlemen of whom Dr. Kenny has just spoken.

President of Congress: I think you are practically proposing an amendment which has already been negatived.

Dr. Hone: No, I am speaking against the amendment. This has been very carefully thought out by the committee, and we do want to keep it in the lines in which it is laid down, so as to gradually bring about this amalgamation.

Professor Allen: If in those two committees there is power to add to their numbers, it gives just the power of homologation that is wanted. It is exactly for that purpose and for no other purpose whatever that I want to have a little elasticity.

President of Congress: I hope that Professor Allen's amendment that "with power to add to their number" be added, will be carried.

On being put to the meeting the amendment was carried.

President of Congress: It now reads:—

> "Membership. Such Commission shall consist of the President, the Honorary General Secretary, the Honorary Treasurer, and two members appointed by the Council of each branch of the British Medical Association, with power to add to their number."

That has been carried. (Cries of "No," and "Yes, it has.") It has been carried by a majority. I will put it again to the meeting.

The amendment, on being put again to the meeting, was, on a show of hands, carried by a large majority.

Dr. GUY GRIFFITHS: I beg to move a further amendment to sub-section (*f*) of section 2; that it be altered to read:—

> "Provided no report shall be offered to any medical newspaper which publishes advertisements of unqualified practitioners."

PRESIDENT OF CONGRESS: Do you observe the words here: "that the President may approve"?

Dr. GUY GRIFFITHS: I wish to limit his approval. (Laughter.) A few days ago I received copies of a medical newspaper published in Australia, which contained advertisements of three or four unqualified oculists.

A MEMBER OF CONGRESS: Spectacle makers.

Dr. GUY GRIFFITHS: They practise as oculists and they are not qualified. That journal is the *Australian Medical Journal*. After all our talk about quacks and quackery I think it is absurd for the Congress to permit such a thing. I think it is a most serious matter. They were advertising as being prepared to measure. You will see the advertisements for yourselves.

Dr. HUGHES: Might I ask if Dr. Griffiths has that paper in his pocket?

A MEMBER OF CONGRESS: You can get it here.

Dr. KENT HUGHES: I only want to explain that by a mistake an advertisement of the character that Dr. Griffiths mentions did appear, and it went out next number.

Dr. GUY GRIFFITHS: There are three of them.

Dr. KENT HUGHES: There is only one where he advertised that he was prepared to measure for spectacles.

PRESIDENT OF CONGRESS: Does anybody second Dr. Griffith's amendment?

The amendment found no seconder.

Dr. HONE: I want to move an amendment to sub-clause (*c*) of section 2, to strike out the words "Commonwealth of Australia," because the carrying of that previous amendment means this: this Standing Commission, as now constituted, supposing the next Congress meets in New Zealand, shall consist of the President, General Secretary, Treasurer, and two representatives of each State. Four of that number forms a quorum. That is in the constitution. You have left no safeguard there, and you might have a large committee meeting composed entirely of New Zealand members who are not competent to advise on matters affecting the Commonwealth of Australia. It does not seem to me a fair thing to put it in that way.

Dr. WORRALL: I can see plainly that the intention of those who drew up the recommendations of this Committee might be absolutely detroyed by the adoption of such an amendment as that proposed by Professor Allen, and I do hope that you will allow us to reconsider it.

PRESIDENT OF CONGRESS: Sub-clause (*c*) reads:—

> "To be of service, as an advisory body in medico-political matters, of the Government of the Commonwealth of Australia and the Dominion of New Zealand."

They would not give their advice if they were not asked. They would only be there if they were asked.

Dr. R. H. Todd: If I may be permitted to say a few words, I have personally taken great interest in this matter, as have all members of the Committee. There were twenty or so of us. We discussed it very carefully from all points of view. We found one of the great difficulties was to apportion the representation and so on, fairly and properly, so as to safeguard the interests of the profession as a whole, as against a meeting of a quorum, so to speak, in any particular State or the Dominion, and so as to keep every meeting that could possibly be held, a meeting of representatives of the whole of the profession. (Hear, hear.) The passing of Professor Allen's motion (we know that he has nothing but goodwill towards the profession, and he has the interests of our work at heart) would be undesirable, and, in this particular instance, I am quite sure that he has made a great mistake. (No, no.) If he thought more of it, or if he had given it the very careful consideration that the members of the Committee have given to it (and let me remind you he was not a member), he would have seen the difficulty which was going to be brought about by the introduction of such a suggestion. We tried to make this committee as small as possible, consistent with its being properly representative, in order that the expense of the matter might be reduced to a minimum. That was one of our great difficulties, and if we have power to add to the numbers it injures the symmetry of the whole committee and puts it in the power of any quorum in any State to go and control the whole matter. Further, I think it would break up the confidence which I hope the profession would have in this committee.

Professor Allen: I do not think it matters very much whether we give this express power or not, seeing that under "(*b*)" we have got this:—"To note the course of medico-political events of importance in Australasia, and, where, in its opinion, intervention by it, as representative of the medical profession in Australasia is urgently called for, to take such action as it may deem appropriate to safeguard the interests of the profession." If those general terms are carried out, it should be sufficient.

Dr. Kenny: (*c*) is merely a re-expression of (*d*).

President of Congress: Do you press your amendment, Dr. Hone?

Dr. Hone: No. I move another one, though,—

"That seven shall be a quorum."

Dr. Worrall: I second that.

Dr. Lendon: Would it meet the case if we provided that the committee shall not meet out of Australia until the year in which Congress is held in New Zealand?

Several Members of Congress: No.

Dr. Butler: That would not do, because the chief men in the committee this year will be the main members of the Congress next time.

President of Congress: Dr. Hone has moved, and Dr. Worrall seconded, that the quorum be altered to seven instead of four, as provided in sub-clause (*b*) of section 2.

Dr. Butler: If you have a quorum of seven you will never get a meeting, because five will be the total number you will get in New Zealand.

Dr. J. R. Purdy (New Zealand): I was going to point out the very same thing, with one little addition,—that raising the quorum to seven would be about the worst thing that could be done, because they would have to appoint probably any three members in New Zealand to get a quorum, and therefore, the very wording of this amendment would be harmful. If we have power to alter our numbers, we might appoint thirty-three.

Dr. NEWLAND: Would it not get over the difficulty by limiting the number to be added by two, and making the quorum five?

Dr. SINCLAIR GILLIES: Might I ask that the committee be requested to reconsider Professor Allen's amendment. I think a good many of the members, after what we have heard, would be inclined to vote differently as regards that proposal, to add to their number.

Dr. NEWLAND: I second that.

Dr. WORRALL: I seconded Dr. Hone's amendment in order to ask that the amendment of Professor Allen, which was carried, might be recommitted. I do not think members fully grasped the possible effects of such an alteration in the original clause as drafted by the committee. It might conceivably result in additions to the Commission of Congress of those who were out of sympathy with the present organisations in each State, namely, the British Medical Association branches, and, as a consequence, action might be taken detrimental to the interests of the profession. Every matter which has been brought up at this Congress for the benefit of the profession has brought us face to face with the paramount necessity of at once improving our organisation if the profession is to even maintain its position. Unless the Congress can be brought into touch with the organisations in each State it is a weakness and danger. I do earnestly hope the vote will be allowed to be taken again.

PRESIDENT OF CONGRESS: It is a little bit out of order; but if the meeting will carry the rescinding of that amendment, there is no objection to it. It was carried by such a very large majority, though.

A MEMBER OF CONGRESS: But they did not understand it.

Professor ALLEN: I have no objection to it being reconsidered at all. I would suggest, as doubt has been cast, we should take another vote; but before that is done, I ask the members seriously to consider just this very simple little thing: we provide that the President, Honorary General Secretary, and Honorary Treasurer of Congress, shall be members of this committee. They will not, in such capacity, be members of the joint committee representing the branches of the British Medical Association. Therefore, I would desire that the Joint Committee of the British Medical Association should have the power to co-opt to themselves from time to time the highest officers of the Congress so as to keep the two bodies working together, and, in like fashion, I should like to have power in the Congress Committee to co-opt to itself anybody it may please, who might be in the other committee, and not in this, so that, as far as possible, the members of the two committees would be identical. That is my purpose.

A MEMBER OF CONGRESS: Limiting the co-option to members of the committees?

Professor ALLEN: Do you think the committees are going to make asses of themselves? Are we not a live body? Do we not know what we are about? Are we going to throw away the main purposes at which we are aiming? If you cannot trust your committee, tear the paper up! (Hear, hear.)

Dr. R. H. TODD: Leave it as it is, then.

Professor ALLEN: I do not want to have it said that my amendment was carried by any misunderstanding whatever. I ask that the amendment be put once again to the meeting to see what the opinion really is.

Dr. SINCLAIR GILLIES: The committee who drew this up was a very representative and by no means small one, and they took quite a long time over these proposals, and if Professor Allen knew the amount of care that has been given to them, I am sure he would change his mind. There were reasons very wise and very grave why it was done.

Dr. HAYWARD: About six hours were given to this question. It was discussed fully by men equal to any here (I will except Professor Allen, of course), and the whole matter was gone into most exhaustively. This was the result—the unanimous result of six hours' solid debate. I ask you to trust the sub-committee that you elected. In their judgment they have done right. I am sure they have done right.

PRESIDENT OF CONGRESS: Somebody has moved that the amendment we carried a little while ago should be rescinded.

Professor ALLEN: Let us assume that we have not considered it.

PRESIDENT OF CONGRESS: A definite resolution has been carried, and we must rescind it before we can go on. It has been moved by Dr. Sinclair Gillies, seconded by Dr. Newland, that the amendment we have already carried, "that the committee shall have power to add to its numbers," shall be rescinded.

The motion was carried.

PRESIDENT OF CONGRESS: Now anyone can move that it be carried as it stands.

Professor ALLEN: This, then, becomes the substantive motion. We have now to vote one way or the other, an opportunity given to us by the rescinding of my amendment. I do not care a button what you do. I only want to do what is right. It seems to me if you want to get those committees running in the way they ought to run, you must have a little elasticity. That is my only argument—absolutely nothing else. Will it help to put these words in, or will it not?

Dr. WORRALL: If the suggested words were added, it may be that results would be achieved which are not representative of our organisation throughout the State.

Dr. HURST: If you do not pass that resolution you restrict the committee absolutely. It is quite possible the committee might wish to have the services of some gentleman who would be extremely useful.

A MEMBER OF CONGRESS: They can do it; they have power.

Dr. CRAGO: As a member of the committee, I should like to say that this received great consideration, and I think the powers asked for are already covered by (*b*). I do hope, now that the amendment has been rescinded, that we shall carry the report as drawn up originally by the sub-committee.

Dr. SINCLAIR GILLIES: I beg to move that the report of the sub-committee be accepted as it is printed.

The motion was carried.

RECOMMENDATIONS OF THE CONTRACT PRACTICE COMMITTEE.

In accordance with the Resolutions passed at the Business Meeting of Congress held on Monday, September 18, 1911, the Contract Practice Committee begs to submit the following Recommendations:—

I. That a copy of the Income Limit Clause passed at the last Session of Congress held in Melbourne in 1908 be sent to the Secretary of the British Medical Association Branch in New Zealand and every Australian State, with an earnest request that steps be taken to enforce the same as soon as possible. The Clause referred to reads as follows:—" That no medical man should pass for admission into any Friendly Society, Lodge, Club, or Association for professional attendance at contract rates of remuneration, any person whose income from all sources, together with that of his wife (if any), exceeds £4 per week, that is, £208 per annum. Further, no medical man should attend professionally at contract rates any member of the abovementioned organisations whose income from all sources, together with that of his wife (if any), exceeds the sum of £6 per week, that is £312 per annum."

II. That in order to facilitate this the first steps to be taken should be:—

(*a*) The strengthening of every Australasian Branch of the British Medical Association.

(*b*) The formation of the nucleus of a fund for the assistance of members who suffer financially through the enforcements of the resolutions of Congress.

III. That the enforcement of the abovementioned resolution be carried out, (*a*) through the Secretary of the Branch of the British Medical Association in each State and New Zealand, (*b*) simultaneously throughout each State, and (*c*) that the provisions of the resolution should not be made retrospective.

IV. That the attention of Congress be directed to the inadequate remuneration received by Medical Officers under contract practice in some parts of Australasia, and that where such inadequate remuneration exists the various Branches of the British Medical Association should take steps to remedy this state of affairs.

V. That no medical man should act as Medical Officer to any proprietary medical aid or benefit Society.

VI. That the remuneration accepted by Medical Officers of Friendly Societies should be upon a " per capita " basis and not by fixed salary.

G. H. ABBOTT, CHAIRMAN.
C. H. E. LAWES, HON. SECRETARY.

Dr. G. H. ABBOTT: Mr. Chairman, Ladies, and Gentlemen,—I wish to bring forward the recommendations of the Contract Practice Committee.

Dr. T. J. DAVIS: Are there any reporters here? I do not think this matter thould be discussed when anybody outside the profession is present.

Dr. G. H. ABBOTT: I do not think we are ashamed of these resolutions, Mr. President, and we want to act fairly to the public as well as to ourselves. (Hear, hear.) I propose that these resolutions be taken as read and adopted.

Dr. HOWARD: I wish to draw attention to some errors in the wording of clause 5. First of all, the ladies have been ignored. (Cries of " Oh !") It means that only a man can act as a medical officer. I suggest that the word " man " be replaced by " practitioner." The latter part of the clause is ambiguous. I have not the wide legal experience of my friend, Professor Allen; but I think it is distinctly misleading.

Dr. G. H. ABBOTT: A comma should be left out, that is all.

Dr. HOWARD: If you leave the comma out, it would be all right; or would you word it in another way: " Proprietary society supplying medical aid or benefit."

Professor ALLEN: Put "proprietary" in twice. Make it "proprietary medical aid or proprietary benefit society."

Dr. HOWARD: That would do. I propose that those verbal alterations be made.

The motion was put to the meeting and carried.

PRESIDENT OF CONGRESS: Clause 5 now reads:—

> "That no practitioner should act as Medical Officer to any proprietary medical aid or proprietary benefit society."

Dr. BINNEY: I have a suggestion to make with regard to section 5 of these recommendations. I noted the error in punctuation. I think that it would serve the purpose if the comma were left out and it read that "no medical man shall act as Medical Officer to any proprietary medical aid or proprietary benefit society unless authorised by the profession." (Cries of "No.") We have in this city a society run on the lines of a medical aid society, which is authorised by the profession, and has been one of the most successful societies we have had. Its offices are in this building. I am connected with it, and I shall be happy to introduce anyone interested in contract practice to the office and show them how it is managed. It is quite possible that we might have to extend that medical society. If we had to do that we would have to conduct medical aid societies. We could not make them, for business reasons, proprietary medical societies, so far as they concern ourselves. I think if we added after "benefit society" the words "unless authorised by the profession," that would allow us to conduct medical societies where we found it necessary for our own protection, and as we have done so successfully here.

PRESIDENT OF CONGRESS: What are the exact words of your amendment?

Dr. BINNEY: Omit the comma after the word "proprietary," making "proprietary" an adjective, and then, after the words "benefit society," put the words,—"unless authorised by the profession." That, I think, would meet the case.

PRESIDENT OF CONGRESS: What do you mean by the "profession"?

Dr. BINNEY: I was using the term as it has been used in connection with our own medical aid society; it was authorised at a meeting of the profession, not the British Medical Association; it was authorised at a general meeting of the profession.

Professor ALLEN: I think some words of that sort would be of service in connection with intermediate hospitals generally. We are not a proprietary body. We are not a court of justice to deal with anybody. This is merely an expression of the opinion of the largest body of the profession.

Dr. KENT HUGHES: Surely that is dangerous! Any three or four men could start a proprietary society under that.

Dr. LOCKHART GIBSON: It is the opinion of the Standing Committee of Congress.

Dr. FETHERSTON (Victoria): I think before next Congress we will have started a benefit society working in conjunction with New South Wales, so that we should have one society for Australia controlled by the profession. That was our idea.

Professor ALLEN: Put it alternatively: "Unless authorised by the Standing Committee of Congress or by the local branch of the British Medical Association."

PRESIDENT OF CONGRESS: Will that do, Dr. Binney?

Dr. BINNEY: Certainly.

> "That no medical man shall act as Medical Officer to any proprietary medical aid or proprietary benefit society unless authorised by the Standing Committee of Congress, or by the local branch of the British Medical Association."

On being put to the meeting, the amendment was carried.

Dr. JAMES BOOTH: I should like to move the addition of the following clause to section 2:—"The establishment of a federation of all the medical defence associations in the Commonwealth and New Zealand," because, when we in Broken Hill were fighting the establishment of a medical institute, members of the New South Wales Medical Union accepted office under the institute. If we had a federation, such a thing would be avoided.

Dr. CLARENCE READ: Our medical union has a different status altogether to your union; ours is for legal defence purposes.

Dr. JAMES BOOTH: There are other medical associations besides those of New South Wales; those of Victoria, South Australia, and Queensland are amalgamating for defence purposes.

PRESIDENT OF CONGRESS: How can you federate associations which exist for entirely different purposes? One is for legal defence. I only throw out the idea.

Dr. R. H. TODD: I think that Dr. Booth's suggestion is outside the scope of this report altogether. There is no necessary connection between any one of the defence unions and contract practice.

Dr. J. MACDONALD GILL: As Secretary of the New South Wales Medical Union, I should like——

PRESIDENT OF CONGRESS: That amendment has not yet been seconded.

Dr. CARRUTHERS: When we give our views from our side of the question, I think you will find that when we come to deal with these people, that there will be a difficulty with regard to clause 6. As I have had some practice for some years in a place where there is a large friendly society, I think it would be better if these words were added at the end:—"Fixed salary without the right of private practice." I think the whole trouble with these friendly societies is that they offer more remuneration with the idea that they pick up private practice. I do not think there is any objection to these friendly societies amalgamating and giving a good fixed salary for a medical man doing their work, provided they take the right of private practice away from him. (Cries of "No.") I think you will agree with me that when we go before the public (and after all we have to take the public slightly into our consideration), they might say: "We give a fixed salary. We employ a man at a fixed salary and the market value of his services." Do not the Government do that at the present moment? Do they not do it as regards the asylums? You will find that the public will probably take that point of view. I only make this suggestion, and I think we ought to be fair in dealing with these societies. If they choose to pay a fixed salary and the market price of a man's services, we have no right to say to a man: "You have no right to work for £500 a year." You might as well turn round to the Lunacy Department, or any other department, and say that the medical men there should not work for the salaries that they do. If you will look at it from a fair point of view, you will see that, with that addition, there will be no harm at all—a fixed salary without the right of private practice. If they get a fixed salary, we would have plenty of medical men applying for these positions.

PRESIDENT OF CONGRESS: Does anybody second that?

Dr. GUY GRIFFITHS: I beg to second that.

Dr. CLARENCE READ: It is the sort of thing that the General Medical Council at home will not permit, for a society to go and employ a medical man and make money out of him. That is the sort of thing that Dr. Carruthers suggests.

Dr. CARRUTHERS: Not at all.

Dr. CLARENCE READ: Friendly societies go and get contributions from their members for medical attendance, and——

PRESIDENT OF CONGRESS: As far as I understand, I think you are under a misapprehension as to what Dr. Carruthers' amendment is.

Dr. CARRUTHERS: At Balmain we have a friendly society dispensary, and there is not a single lodge benefit.

PRESIDENT OF CONGRESS: As far as I understand Dr. Carruthers' amendment it is simply to prevent a man taking a lodge at a fixed salary with the right of private practice as well. He would prohibit a man taking a lodge at a fixed salary and at the same time preserving to himself the right of private practice as well. Is that correct, Dr. Carruthers?

Dr. CARRUTHERS: That is exactly what I meant, Sir.

Dr. CLARENCE READ: The friendly society members pay into their society contributions for medical attendance and medicine, and under this system that Dr. Carruthers suggests, they will pay a man perhaps £400 or £500 a year without the right of private practice. They might pay him £600 a year. We have only past experience to go by. We know that they will sweat the poor devils. Look at that institute at Brisbane. There was a medical man who held the position of medical officer at a paid salary, and he said: "Doctor, I would sooner go and break stones on the road than do that sort of thing." I do not wish to detain the meeting. I only wish to emphasise how wicked a thing it would be to pass that amendment.

PRESIDENT OF CONGRESS: It is for the meeting to vote against it or for it.

The amendment, on being put to the meeting, was negatived.

PRESIDENT OF CONGRESS: The 5th clause now reads:—

> "That no medical practitioner shall act as Medical Officer to any proprietary medical aid or proprietary benefit society unless authorised by the Standing Committee of Congress or the local branch of the British Medical Association."

With that one amendment I submit all these resolutions from the Contract Practice Committee.

Dr. KENNY: Might I ask one question. There is not the least doubt that the committee have gone into this matter most thoroughly. I merely want to know this for my own information's sake. The rate of income for admission of new members into the friendly societies is £4 per week throughout. Are we to assume from that that the committee have taken into consideration the £4 single man and the £4 per week for a man with a large family?

PRESIDENT OF CONGRESS: I cannot answer for what is in the mind of the committee. These resolutions were passed at the last Congress. That is a quotation from the resolutions of last Congress.

On being submitted to the meeting, clause 5, as amended, was carried.

PRESIDENT OF CONGRESS: I should like to say that the number who have joined this Congress is 906; 543 in attendance. (Applause.)

The next business is the fixing of the committee for the next Congress.

Dr. WORRALL: I have a formal resolution. I move that the Hospital Abuses Committee appointed at the last Congress be reappointed, viz.:—

> President, Honorary Secretary, Honorary Treasurer; Dr. J. J. Holland, Dr. A. J. H. Saw, Dr. W. Pope Seed, Western Australia; Dr. Ewart, Dr. E. Roberton, New Zealand; Dr. R. Worrall, Dr. C. B. Blackburn, New South Wales; Dr. W. R. Cavenagh-Mainwaring, Dr. W. T. Hayward, South Australia; Dr. J. Lockhart Gibson, Dr. J. B. McLean, Queensland; Dr. G. H. Butler, Dr. Pardey, Tasmania; Dr. J. W. Barrett, Dr. Lewers, Victoria. Dr. Worrall, Convener.

Dr. J. W. BARRETT: I second that.

The motion was carried unanimously.

PLACE OF MEETING OF NEXT SESSION OF CONGRESS.

Dr. VALINTINE (Accredited representative of the Dominion of New Zealand): It would be very difficult, if not impossible, for us to extend to visitors the same hospitality that has been accorded to us here. Nevertheless, if we should have the benefit of a visit to our poor islands we will promise visitors a right royal welcome, and we will do our utmost to make their stay in New Zealand an enjoyable one in every way. I have pleasure in moving accordingly.

PRESIDENT OF CONGRESS: Any approximate time?

Dr. VALINTINE: Some time in 1914; we will not state any month.

PRESIDENT OF CONGRESS: It would be better not to commit ourselves definitely just now as to any particular month. Although this comes in the form of an invitation, it has to be carried in the usual way. Say in or about 1914.

Dr. SEYMOUR BREWIS: I have much pleasure in seconding that.

Carried unanimously.

PRESIDENT OF NEXT CONGRESS.

Dr. Hope Lewis: I beg to propose Dr. Arthur Challinor Purchas as the President of the next Congress to be held in Auckland some time in 1914.

Dr. Davenport: I have very much pleasure in supporting Dr. Hope Lewis's motion. I have known Dr. Purchas for some years as a student in Edinburgh, and there was no better sport, no better comrade, and there is not a better man in the profession to carry the heavy load that has been left him to carry. I am sure every member of the Congress will bear me out when I say that he will support the burden ably.

Carried, with acclamation.

President of Congress: Dr. Purchas is my oldest friend in the profession, and it is rather a coincidence that two successive presidents should be men who have lived in the same rooms in Edinburgh. We "dug" together in Edinburgh, and we were in Vienna together. It is rather curious that we should come together in this way.

Dr. A. C. Purchas: I feel very humble-minded at the present moment. I feel I am undertaking a task which is beyond me; but when I look round the room and see quite a number of my old fellow-students, men who backed me up in difficulties in the past, men who cheered me on and encouraged me, I feel that I must make an effort, and, at any rate, do my best. Also I feel that I would like now personally to urge every one of you, my old friends and my new friends (because I have made quite a number of new friends since staying in this hospitable city of yours), to come across in a body—in one ship if you can, because it is very much more sociable. Do not worry about the horrible voyage which everybody talks about; it is not so bad when once you get on board. Those who can must come in their airships. I hope every member of this Congress will endeavour to come to Auckland. What we cannot do in the form of large, magnificent, and brilliant entertaining, we will try to do in the way of natural wonders, in the most hearty manner possible. I have therefore very much pleasure in thanking you for the very high honour which you have conferred upon me, and also to extend the warmest possible invitation to the meeting in Auckland in 1914.

INTERNATIONAL CONGRESS OF MEDICINE.

President of Congress: The International Congress of Medicine, meeting in London in 1913, have asked this Congress to nominate men whom we consider eligible for Vice-Presidents, &c., of the various sections. That is a thing we cannot do now, and I think I might formally move that this matter be referred to our Standing Committee. Is that carried?

Carried unanimously.

Dr. Worrall: I have a formal motion:—

> "That copies of the speeches delivered at the General Meeting on Monday last, with regard to hospital abuses, together with the resolutions that have been passed, be sent to all branches of the British Medical Association throughout Australasia."

Carried unanimously.

VOTES OF THANKS.

President of Congress: Now I am going to ask some votes of thanks to be passed.

Dr. Lockhart Gibson: I have been asked to move the following vote of thanks, and I do it with great pleasure:—

> "That the Congress tender to His Excellency the Governor-General its grateful thanks for opening the Session, and expresses its appreciation of the interest that His Excellency and Lady Denman have taken in its proceedings."

Carried, with acclamation.

Dr. G. A. Syme: I move:—

> "That this Congress tender to His Excellency and Lady Cullen its thanks for their hospitality to members of Congress and their wives."

Carried, with acclamation.

Dr. Haywood: I move:—

> "That this Congress tender to the Federal Government its thanks for its countenance and support, especially with regard to the bringing of the filarial patients from Queensland."

Carried, with acclamation.

Dr. Clendinnen: The honour has been conferred upon me of moving:—

> "That the thanks of the Congress be rendered to the State Government of New South Wales for its countenance and support, particularly with regard to the Harbour excursion, the trip to the Jenolan Caves, and the visit to Waterfall."

Carried, with acclamation.

Dr. Foreman: I have much pleasure in moving:—

> "That a vote of thanks be passed to the Honourable the Premier (J. S. T. McGowen, Esq.), for his kindness in being present at the Inaugural Meeting and welcoming the members; to the Railway Commissioners of the various States and the Dominion of New Zealand."

Carried, with acclamation.

Dr. S. H. MacCulloch: It is my pleasure to move:—

> "That the thanks of the Congress be accorded to the Lord Mayor for his reception and for attending and speaking at the Inaugural Meeting."

Carried, with acclamation.

Dr. Butler: I move:—

> "That this Congress expresses its great indebtedness to the Chancellor, Vice-Chancellor, and Senate of the University of Sydney, for the grant of the use of the University buildings and grounds. To the Professors and lecturers who placed their rooms at the disposal of the Executive. To the Registrar, Mr. Barff, and his staff for their kind assistance. That this Congress warmly thanks the President and Council of the New South Wales branch of the British Medical Association for the use of the library for meetings, and their hospitality to the members of Congress and their wives. And to the authorities of the various hospitals and institutions that invited inspection and facilitated the work of the Congress."

Carried, with acclamation.

Dr. NEWLAND: I have pleasure in moving:—

> "That a special vote of thanks be accorded to Mrs. Pockley, President of the Ladies' Committee, Mrs. W. G. Armstrong, Honorary Secretary, and Mrs. Bowker, Honorary Treasurer, and the members of the Ladies' Committee, for their splendid hospitality to the members of Congress and the ladies accompanying them."

Carried, with acclamation.

Dr. HONE: I move:—

> "That this Congress expresses its keen appreciation of the hospitality shown to its officers and members and their wives by the Royal Army Medical Corps, Miss Walker and the trustees of the Walker Hospital, Australian and University Clubs, the Automobile Club, the Royal Sydney, Kensington, Killara, and Manly Golf Clubs, the Australian Jockey Club, the Ladies' Patriotic Club, the Women's Club, the Trustees of the R. T. Hall Sanitorium, Sir Philip Sydney Jones, Dr. McIntyre Sinclair, Sir James Fairfax, the President and Committee of the Royal Yacht Club, the Directors of the Sydney Ferries, and all those ladies and gentlemen within and without the profession who have so kindly entertained members of Congress and the ladies accompanying them."

There is a personal note in the end of that resolution which I think appeals to us all, at any rate to all of us who are visitors from other States. One of my pleasantest recollections of the Adelaide Congress is the graceful appreciatory remarks uttered by the late lamented Dr. Thomson when proposing a vote of thanks like this for the hospitality that had been extended to the visitors there. Well, all I can say to Sydney is: "Go on top." We cannot in words express our sense of the appreciation that we feel is due to the medical men of Sydney and their wives and other individuals who have helped to make this Congress such a tremendous success. It is not merely a matter of form; it is not merely from the social side that I say this. A great deal of the help we receive from Congresses arises through our intercourse with our professional brethren. Might I single out (because it is the only opportunity I will have, personally) the President, and express to him our deep sense of appreciation of his worthy efforts.

Received with acclamation; members rising in their seats and singing: "For He's a Jolly Good Fellow."

Professor ANDERSON STUART: I move:—

> "That a vote of thanks be accorded to the following,—the Archbishop of Sydney, the Chapter of St. Andrew's, Archbishop Kelly and the Chapter of St. Mary's, the Presbyterian, the Wesleyan, and Congregational Churches, and the several preachers for the special Congress services on Sunday, 17th September; to the Premier and Government of New South Wales for undertaking to print the transactions of Congress; to the Postmaster-General for providing postal, telegraph, and telephone services at the Medical School."

Carried, with acclamation.

Dr. G. H. Abbott: I have much pleasure in proposing a hearty vote of thanks to Dr. Palmer, Dr. MacCulloch, Dr. Brady. I am sure we are all very grateful to them for the splendid work they have done towards making this Congress such a success. To Dr. Palmer we are especially indebted. You all know how energetic he has been; but only those in intimate contact with him can understand the immense amount of work he has put into this Congress.

President of Congress: I ask you to carry that with "three times three." Nobody knows more than the President what these men have done.

Three cheers were given.

Dr. Palmer: Mr. President, Ladies, and Gentlemen,—I will not detain you any longer. I thank you. I know you will overlook any little blunders that may have been made. You know that we have done the best we could. Really, most of the work, or a great portion of it, has been upon the shoulders of the President; but you will hear more about that afterwards. I can only thank you very much for the kind way you have received me.

Dr. MacCulloch: Mr. President, Ladies, and Gentlemen,—I have done very little for the Congress, except collect money and prevent my colleagues spending it. I thank you.

President of Congress: Dr. Todd has a resolution which I know you will receive heartily.

Dr. R. H. Todd: It is my privilege to vote our special thanks to our friend, Mr. A. W. Green, who has been the lay secretary of the Congress for this Session. To those members who live in New South Wales this motion of mine requires no elaboration from me; but to those who live outside New South Wales I might say, in the shortest possible words, that this Congress has been aided enormously by the intense goodwill, the wonderful patience, and the great experience of our friend, Mr. A. W. Green.

President of Congress: I put that to the meeting, and I also ask you for three cheers for Mr. Green.

Three cheers were given.

Mr. A. W. Green: Ladies and Gentlemen, I thank you heartily.

Professor Allen: Ladies and Gentlemen,—It is my great privilege this morning to propose the most hearty vote of thanks we can possibly give to our President for all his services to the Congress. We know something of the big labour involved, from past Congresses. We have seen its activity in our own States, and we have heard of all the work that has taken place in Sydney. But we only know a little of the actual labour he has given to make this Congress of vast interest to medical science throughout Australasia, to provide in every way for our comfort at these meetings, and to see that our deliberations shall be productive of the greatest good to the profession and to the public. We know only a little of these things, but we have been able, to a certain extent, to see our President's activities throughout the progress of the Congress itself, and I am sure we recognise, with the deepest thankfulness, all he has done from the moment of our first coming to Sydney, to this time—

right on to the hour when the proceedings of Congress are being closed. We know with what dignity, with what practical wisdom he has presided over our deliberations; how he has helped to guide us in right ways, so that, in striving to obtain good in the future, we may not fall into harm. We have already thanked him for the splendid address at our Inaugural Meeting in the Town Hall. We now wish also to thank him for the great personal kindness he has shown to us. We wish him to recognise that we appreciate to the full the splendid hospitality we received at the beautiful party the other night, and to couple with our thanks to him our gratefulness to Mrs. Pockley, who added so much to our pleasure on that memorable occasion. (Loud applause.) If the President has any hesitation (as he told us at the meeting he had) with regard to his fitness for the Presidency, let us tell him now that we have no doubt at all in the matter, and we simply congratulate him on the glorious success of all his endeavours for the good of this Congress. We wish him, and Mrs. Pockley, and their family, long life, all good health, all the joy, all the good things that life can bring them, and we say to them,—Thank you. Well done, well done!

After hearty cheering, the members rose in their seats and sang: "He's a Jolly Good Fellow."

President of Congress: Professor Allen, Ladies, and Gentlemen,—I knew I should have to face this ordeal, and I scarcely know how to thank you for the very kind words that have been spoken. You have completely overwhelmed me, and I do not feel that I can thank you sufficiently. In Melbourne, you may remember, I told you that I would do my best. I have tried to do my best. (" You have exceeded it.") I also told you that Mrs. Pockley would do her best, and she has done it in so far as her frail health would allow her. The chief anxiety I have had (and perhaps you will understand, although not realise it), has been for her, as to how she would be able to go through the ordeal. I am sure you will be glad to know that she has come through all right (Hear, hear) although she has had to forego the pleasure of attending a few of the social functions. I also have to say that the tribute that Professor Allen has given to me is the sort of thing one hears on these occasions. (No, no.) (" You deserve it.") I thank you very much for the sympathetic way you received his remarks; but everybody knows that the work of the Congress is done mostly by the Secretary and Treasurer. (" And the President.") I am here just to put in a word now and again, and to approve, or disapprove, as the case may be, of what they have done. (Oh, no.) I am glad to say I have never had anything to disapprove of. We have a most harmonious committee. We have never had the slightest friction—not the slightest hitch. Everything has been carried unanimously from the very beginning up till now—the end. It has been said that I must be glad it is all over, or that I will be glad when it is all over. I am glad and I am not glad. I am glad that it is over without a hitch, or without any serious hitch, and that my anxiety in that way is relieved. I am sorry, because I shall be saying good-bye to many old friends, and not a few new ones, who, possibly—many of them at least—one may never see again; and also, during the preliminary work of Congress on the Committees, it has been my privilege and advantage to come into closer touch with many men in the profession with whom I had only a slight acquaintance before. I may say a more splendid lot of fellows all round I have never had anything to do with. It is one of the great pleasures in a work of this kind to find, very often, qualities which you do not dream of or suspect until you come into contact with men in the way I have come into contact with these men. And the way they have nursed me during the past week, and looked after me with their tender solicitude, has affected me very much. They have nursed me with the greatest care. I have to apologise to one or two men for going off at a tangent, and occasionally losing my temper under the rather great strain. Professor Allen knows, as well as I do, and Dr. Butler also, the strain that there is on the President. I do not want to make it out to be any greater than it is; but only those who have had the Presidentship have any idea what it is. Occasionally I have said things to one or two members of Congress for which I am sorry, and for which I should like to apologise. On behalf of my wife, I thank you. As I say, she has done her best ("And done it well"), and she will be very grateful to receive your cordial expressions of approval.

A vote of thanks was given to the Press, moved by Dr. Dick, and the proceedings terminated.

SECTIONAL DINNERS.

The sectional dinner of the sections of medicine, anatomy, and physiology, public health, pathology, bacteriology, and tropical medicine took place at the Hotel Wentworth. About ninety-six members of Congress took part. Sir Philip Sydney Jones filled the chair, and proposed the toast of "The King." "The Visitors" was proposed by Dr. Scot Skirving, and was replied to by Professors Allen and Berry and Drs. Hayward, Wilton Love, and J. S. Purdy. Sir James Graham proposed the toast of "The Chairman."

The sectional dinner for surgery, including the naval and military section, was held at Aaron's Exchange Hotel on the evening of Wednesday, 20th September, 1911. Dr. Fiaschi was in the chair. After the usual toast of "The King," that of "The Visitors" was responded to by Mr. Syme, of Victoria; Colonel Purdy, of New Zealand; the Hon. G. H. Butler, of Tasmania; Dr. Giles, of South Australia; and Dr. Freshney, of Queensland. The toast of "The Chairman" was proposed by Dr. Sinclair-Finlay, Balmain. In all, about 130 sat down, and more than half were visitors from the Dominion of New Zealand and other States of the Commonwealth.

The sections of ophthalmology, neurology, otology, diseases of children, radiology, and dermatology, dined at Paris House, 116 being present. Dr. Brady was in the chair. Dr. Clubbe proposed the toast of "The Visitors," and the President of each of the sections replied, viz., Drs. Barrett, Beattie Smith, Robertson, Jeffreys Wood, Clendinnen, and Herman Lawrence. The toast of "The Chairman" was proposed by Dr. A. L. Kenny.

The section of gynæcology dined at the Australian Club to the number of ninety. Dr. Joseph Foreman was in the chair. The toast of "The Visitors" was proposed and responded to by the representatives from the various States.

HARVEIAN THEATRE, MEDICAL SCHOOL.

WEDNESDAY, 20TH SEPTEMBER, 1911.

DISCUSSION ON VACCINES AND SERUM THERAPY.

The Opening Address, by H. B. ALLEN, M.D., Professor of Pathology in the University of Melbourne (President of Congress presiding):—

Jennerian Vaccination.

The foundations of our knowledge of vaccines were laid by Jenner, towards the end of the eighteenth century, and any discussion of vaccines would be imperfect if it did not touch the present position of the Jennerian prophylaxis of smallpox. In Germany, vaccination and revaccination are compulsory, and smallpox has practically been stamped out. For the ten years ending 1908, in a population of sixty-two millions, the greatest number of deaths in any year was sixty-five. For the admission of cases, Berlin now relies on a pavilion of twelve beds off one of the general hospitals.(1) A startling demonstration recently occurred close to Australia. At Caluya, an isolated island in the Philippines, with a population of 2,000, a case of smallpox was introduced. A thousand cases with 400 deaths followed before help came. Two hundred of the inhabitants ran away; 800 were vaccinated. Not one case occurred among those of the 800 who had a successful incubation two weeks old. There were no deaths.(2) In the United Kingdom, however, a system of exemptions began in 1898, and has steadily increased till, in the first half of 1910, out of 456,533 births, 110,851, or nearly 25 per cent. received exemption.(1) In Victoria, about 32 per cent. of infants are not vaccinated in the first year, though the percentage shows little tendency to change. About 365,000 out of a population of 1,315,000 are unvaccinated. There is practically no revaccination, so that it is estimated(3) that 98 per cent. of the population above the age of 10 are liable to attack. It would be well to adopt the suggestion made in the House of Commons(1) that a leaflet setting out the advantages of vaccination be distributed by the Registrars of Births with the usual formal notice; and, above all, the Quarantine Department of the Commonwealth and the State Health Departments should see to it that there is a sufficient supply of pure calf lymph, glycerinated and bacteriologically tested, that vaccination is done solely with such lymph, and that for emergency there is in readiness the means whereby a large stock of pure lymph may be rapidly prepared by the chloroform process.

Pasteurian Vaccination.

The second great step in the history of vaccines followed shortly after Koch's invention in 1876, of the method of growing bacteria on solid media, and the formulation of his postulates for the proof of the causal relation of micro-organisms to disease. Most of the great discoveries of this period are associated with the glorious memory of Pasteur. Time will only permit me to mention two of them. Since the introduction in 1881 of the use of cultures attenuated by heat as a vaccine against anthrax, millions of animals have been protected. In Victoria, during the past three years, fourteen outbreaks of anthrax have been dealt with, and the results are shown in the subjoined table:—(4)

Number of Outbreaks.	Deaths before Inoculation.			Number Inoculated.				Deaths after Inoculation.
23 Dec., 1908, to 30 June, 1911.	Horses.	Cattle.	Sheep.	Horses.	Cattle.	Sheep.	Goats.	
14	56	21	40	1,374	390	507	47	12
Totals	117			2,318				12

Of the twelve deaths after inoculation, fully half occurred within three days, so that immunity had not time to develop. These results, though on a small scale, must be deemed highly satisfactory.

The other great discovery of Pasteur that I desire to mention is vaccination against hydrophobia, by injection of emulsions made from infected spinal cords attenuated by rapid drying. It is now known that an equally good vaccine may be prepared by simply diluting the emulsion of virulent cord. In 1910, at the Pasteur Institute in Paris, 401 bitten persons were treated, including 98 cases in which the presence of rabies in the biting animal was experimentally proved, 143 cases in which it was diagnosed by veterinary examination, and 160 cases in which it was suspected. There was not a single death.(5) Hitherto, distance and a rapid system of animal quarantine have kept Australia completely free from hydrophobia; but we must not count too much on a continuance of this freedom. In New York State, during 1908–09–10, 105 persons were bitten and twelve died. The disease spread into part of Ontario in 1910. It is out of the question to maintain a succession of rabid animals in Australia to furnish the vaccine. The results of passive immunisation by serotherapy are not equal to those of active immunisation. The recent experiments of Sir David Semple,(6) using virus killed by incubation with 1 per cent. carbolic acid, may provide an easy way of maintaining a stock of vaccine for any contingency. Quite recently it has been discovered that poliomyelitis anterior acuta is due, like hydrophobia, to an invisible filtrable virus, and is transmissible to the chimpanzee. The spinal cord dried *in vacuo* over sulphuric acid is virulent for fourteen days, and vaccination with the dried cord is said to give immunity.(7) So far, however, this discovery does not give assistance in treatment. Haffkine's preventive vaccination against cholera consists in the inoculation of attenuated cultures followed by inoculation of virulent cultures. It is therefore comparable with Pasteur's vaccination against anthrax. The method seems to have been of decided service in British India, though Metchnikoff(17) has again directed a sceptical criticism upon it. The recent experiments of Choukevitch(17) on baby rabbits indicate that it does not immunise them against intestinal cholera.

Serotherapy.

If we pass by for the present Koch's researches with tuberculin, the third great development came in 1890 and 1891, with Behring's discovery of the antitoxins for diphtheria and tetanus, followed by Bordet's work with hæmolysins and the brilliant researches and speculations of Ehrlich. In the diphtheria pavilion of the Hôpital des Enfants Malades in Paris, for the four years (1890–93) preceding the adoption of serotherapy, the average mortality was 51·71 per cent. With the use of antitoxin, there was a fall at once to less than one half, and in 1904–5–6, the mortality rates were 7·6, 10·11, and 10·45 per cent., or, if cases proving fatal within 48 hours of admission are excluded, 5·47, 6·97, and 6·4 per cent.(8) Dr. C. V. Mackay, of Melbourne, has drawn attention to the advocacy of large doses by McCollom, of Boston, confirming it by his own experience, in which the average dosage for 400 cases was 12,000 units.(9) By precipitating a large part of the globulin from the serum, Gibson has obtained a purified concentrated antitoxin, not so apt to cause eruptions and other unpleasant phenomena which so often follow injections of horse serum with or without antitoxin.(10) Much smaller doses (500 units) suffice for preventive inoculation, and the question has arisen whether, when the hypodermic method is disliked, preventive doses may not be given by the mouth. McClintock and King, after careful experiments on guinea-pigs, recommend the following procedure in children, so as to avoid the needle puncture and any serum sickness. Half an hour before giving the serum (or dry antitoxin dissolved in water) give a glass of 1 per cent. sodium bicarbonate. When the antitoxin is given, add one minim of fluid extract of opium, and from 4 to 10 minims of saturated solution of salol in chloroform. When possible, give no food for four hours before the serum.(11) In curative work, subcutaneous injection should always be used. Much difficulty has been experienced in the treatment of diphtheria carriers. The creation of active immunity by repeated injection of dead bacilli is tedious. Wassermann and Lipstein have recommended injections of *bactericidal* serum. Sacquepée, analysing the various methods, finds that *antitoxic* serum is of no service, and recommends Louis Martin's method of applying dried *anti-bacterial* serum locally. It is said that, with the use of the Martin pastilles, the bacilli in healthy carriers disappear in eight days.(12)

The results with tetanus antitoxin have not been so satisfactory as those obtained in diphtheria. Apart from diphtheria and tetanus, bacteria act chiefly by their endotoxins, and it is only in very recent times that a satisfactory method has been devised of preparing anti-endotoxins. Besredka discovered that while bacteria introduced under the skin provoke the production of bacteriolytic substances, those introduced directly into the blood cause the formation of definite anti-endotoxins. Studying typhoid fever, Pfeiffer and Bessan found the earlier anti-endotoxic serum of Macfadyen almost without effect, while that of Besredka, injected with the bacilli, clearly hindered in some measure the typhoid intoxication, though absolute neutralisation was never attained.(13) With the adoption of this new way, especially in combination with older methods, serotherapy may enter on a new era. Time will permit only the briefest reference to the results already achieved, whether with anti-bacterial or antitoxin sera.

The most widely known of the sera is anti-streptococcic serum, including Marmorek's serum, obtained by inoculation of a single virulent strain after special culture, and the various polyvalent sera obtained by inoculating mixtures of many strains. Marmorek, Aronson, and Neuberg uphold the unity of streptococci, and claim that their respective sera are effective against all forms; but the variations of streptococci in appearance and in fermentative and hæmolytic powers are associated with differences in agglutination and in toxic effects; so that the polyvalent sera are most in favour. Their value has been tested mainly in puerperal fever and other forms of acute septicæmia; rapid amelioration of symptoms frequently follows, though the mode of action is by no means clear. The vogue of these sera increases year by year, but the whole question is too intricate to permit a clear verdict on their efficacy. Other anti-streptococcic sera have been directed against complications of scarlatina, against chronic rheumatism, &c., but the results are still *sub judice*. Similarly the pneumococcal sera of Klemperer and others have so far not produced decisive results.

In epidemic cerebro-spinal meningitis, according to Wassermann and Leber, the specific anti-serum was unsatisfactory while used subcutaneously, but far greater success was attained when the serum was injected into the spinal canal. In the Rhine Provinces, the mortality was reduced in this way from nearly 80 per cent. to between 12 and 15 per cent., the improvement after inoculation being often instantaneous.(14) With children under two, it is very important that the serum should be used early, as experience in New York clearly showed.(15) The anti-typhoid serum of Chantemesse, though not definitely bactericidal in vitro, causes leucocytosis and rise of opsonic index, and in 1,000 cases of typhoid, Chantemesse had a death-rate of 4·3 per cent., when the other hospitals in Paris had a mortality of 17 per cent. Vaillard and Dopter claim that in the worst cases of bacterial dysentery, their serum has reduced the mortality by three-fourths.(16) In plague, Yersin's serum is the only remedy from which much can be expected, injected into the veins in severe cases, and under the skin in mild cases; but, at the Pekin Conference, it was stated that the results in pneumonic plague were disappointing. As regards cholera, in view of the doubtful efficacy of Haffkine's vaccine,(17) an anti-serum has been prepared by the use of cholera toxin, and is said to be preventive and curative in baby rabbits; but its powers in man have yet to be proved. Calmette's anti-venin is effective against cobra poisoning, when used early, but he is careful to state that a polyvalent anti-serum for the special venoms of Australian snakes should be prepared in Australian laboratories.(18)

Serum Sickness and Anaphylaxis.

The injection of serum in moderate amount is followed in 20 or 30 per cent. by some degree of serum sickness, characterised by local reaction, various erythematous and urticarial eruptions, pains in the joints, more or less remittent fever, and perhaps a trace of albumen in the urine. The second week is the most common time for the appearance of such symptoms, which disappear after a few days. Much more rarely, profound shock occurs. These phenomena may follow a single injection, and are due to a peculiar sensitiveness of the tissue elements, and above all of the nerve cells, to the influence of horse serum. They are reactions against the foreign proteins of the serum, not against the antibodies contained in it. Many authorities incline to treat them as varieties of anaphylaxis, which is also a reaction against foreign protein. In anaphylaxis, however, two injections are necessary. The first, which may be very minute, establishes in ten or twelve days a condition of hypersensitiveness. The second, which must be above a certain minimum, causes, in small laboratory animals, excitement, respiratory distress, paralysis and coma, with great fall of blood pressure, disappearance of all antibodies, and very often speedy death. If the second injection is sub-minimal, or if it is given before the hypersensitive state is developed, there is no shock, and a refractory state of anti-anaphylaxis is established. I can only refer you to Vaughan's experiments on the splitting of proteins,(19) Richet's work on Apotoxin, Besredka's series of memoirs on Sensibilism and Anti-sensibilism in the Pasteur Annales, and to the researches of Friedberger on Anaphylotoxin in many numbers of the Zeitschrift fur Immunitatsforschung. An excellent summary is contained in Chapter XIX of Hiss and Zinsser's Text-book of Bacteriology. A passive state of sensitisation may be created by injection of serum of sensitised animals; and in some cases it is possible to cause anaphylactic shock by injecting a mixture *in vitro* of the sensitised serum and the antigen. It is generally believed that an antigen, in the serum first injected, produces a specific antibody, which combines with the same or another antigen in the second injection to produce the anaphylactic poison.

When two or more injections of serum are given at intervals of at least twelve days, serum sickness may occur in modified form. There may be an *immediate* or *speedy* reaction, with severe local œdema, and possibly erythematous rashes and fever, soon passing off; or there may be an *accelerated* reaction of similar character before the end of the first week setting in abruptly and not lasting long. The immediate reaction and the accelerated reaction may occur in succession in the same patient. Von Pirguet has found a like double response in re-vaccination against smallpox.(20) These phenomena form another reason for including serum sickness with anaphylaxis under the wide group of *Hypersensitive States*.

Wright's Vaccine Therapy.

The latest and most far-reaching development of Vaccine Therapy has its centre in the researches of Sir Almroth Wright, once Demonstrator of Physiology in the University of Sydney (1889–91), afterwards Professor of Pathology at Netley, and now Director of the Department for Therapeutic Immunisation at St. Mary's Hospital, London. Pfeiffer having found in man a development of agglutinins in response to injection of heated typhoid culture, Wright proceeded to elaborate a method of vaccination against typhoid fever by inoculation of cultures sterilised by heating to 60 degrees C. Experiments on a large scale were made in connection with the British Army in South Africa, and also in British India. The technique has been gradually improved, and the results have been more and more favourable, till, in 1910, American statistics showed that among over 14,000 persons inoculated the incidence of typhoid was fifteen times less than among those not inoculated. The War Department of the United States has therefore made inoculation compulsory for all employés in the sanitary service of the hospitals, and for all troops at manœuvres when the drinking water is bad.(21) A negative phase follows injection, so that vaccination should be avoided while the troops are actually exposed to infection. As to the treatment of typhoid by vaccines, experience is small, but Sir William Leishman records some encouraging results.(22)

The next great achievement of Wright was based on Leishman's observation that the plasma of blood had a varying power to encourage phagocytosis. Working with serum, Wright established the existence and powers of opsonins, and built up the whole method of opsonic control of vaccination.(23) A valuable discussion of the subject occurred in 1910, before the Royal Society of Medicine, and has been published under the title of "Vaccine Therapy, its Administration, Value, and Limitations." Excellent descriptions of the technique and analysis of results are available in various handbooks, so that I shall only refer to a few aspects of this vast question. Wright carefully points out that the opsonic test is only a partial test. In addition to opsonins, there are many other factors in immunity. Our knowledge of antibodies is still very imperfect. We know that there are differences between the opsonins of natural immunity and those of acquired immunity. The acquired opsonins are more thermostable and more specific than the normal opsonins, in these respects approaching close to amboceptors. But we can only speculate concerning their precise nature and mode of action. Nevertheless, by careful opsonic estimations, Wright and his followers have vastly extended the scope of vaccine therapy, and have brought accuracy and security into practice formerly governed largely by analogy and surmise. For instance, in the treatment of local staphylococcal infections, the doses and times of administration have been so standardised that Wright finds opsonic control usually unnecessary. It must not be expected that every case of local suppuration will yield to vaccines. The stock vaccine may fail when an autogenous vaccine will succeed. The surgeon must still evacuate pus, and special local treatment may be necessary to promote the flow of antibodies through the affected part. Even autogenous vaccines may fail, or succeed only after unexplained delay. The opsonic reaction may be slower than usual, so that satisfactory results are obtained only when the injections are spaced out much more widely than usual. The index may reveal special susceptibility or relative insusceptibility, so that the dose must be modified accordingly. In the totality, the success is undeniable, and even those who do not use the opsonic index must still be guided by the rules that Wright has formulated in accordance with it. The utility of vaccines is more marked in the chronic and local forms of infection than in the acute and general. Intractable gonorrhœal rheumatism and chronic vulvo-vaginitis often subside in a most gratifying manner, while most observers find that the acute primary disease is comparatively little influenced. Astonishing results may accrue, as when the vaccine treatment of an abscess in an apparent diabetic not only gives protection from the coccal infection but also causes the glycosuria to disappear. The possible realm of vaccine therapy is as wide as the relation of bacteria to disease.

Two great boons conferred by the opsonic method deserve special mention. Firstly may be placed the better understanding, measurement, and control of the negative phase. Secondly, when many bacteria are present, we have a new aid in estimating their true relation to the disease, so that harmless or unimportant bacteria may be disregarded; and where there is actual mixed infection, we have a mode of immunising against the several operating microbes in careful succession and not in disorder.

As to the reliability of the opsonic index, there is no doubt that, in the hands of Wright and of competent observers trained by him, a close approximation to correctness can be obtained. The results in different well-organised laboratories vary when compared with one another, but these in the same laboratory are usually consistent, so that a fall or rise in the index may be accepted. Allowance must be made for the use of different strains of micro-organisms of different ages, for varying presence of agglutinins, for various amounts of care in making the emulsions, for varying degrees of damage done to leucocytes in preparation, and for some variation in the phagocytic power of leucocytes independent of opsonins. The operations are tedious at the best. An hour may sometimes be required to make a proper emulsion of tubercle bacilli. In babies, the indices against tubercle and pyogenic bacteria are very variable, and are often low when the general health and the apparent resistances are quite satisfactory. As to amateur immunisers, with little knowledge of bacteriology, doing the work hastily, rashly declaring the index, and guessing the dose of vaccine to be administered, it can only be said that their results are worthless and their practice full of danger to the patient.

Vaccine Therapy in Tuberculosis.

The difficulties that may be encountered in vaccine therapy are best exemplified by a study of the results obtained in the treatment of tuberculosis. Before announcing his discovery of tuberculin, Koch definitely proved that laboratory animals might be made immune to tubercle by repeated inoculations of tuberculin; and though the early results of its administration to man were unfavourable, there has been a steadily growing conviction that tuberculin is a valuable remedy if properly used. The general decrease of the death-rate from tuberculosis has, I need hardly say, been independent of any new modes of treatment: for example, when the death-rate from phthisis in Melbourne and its suburbs per 10,000 fell from 22·05 in 1885–89 to 11·33 in 1905–09. Similarly, the marvellous results obtained by Sir Anthony Bowlby in 900 cases of tuberculous disease of the hip at the Alexandra Hospital in Bloomsbury were independent of any specific remedies.(24) Belief, however, is now fairly general that tuberculin renders decided service in the treatment of tubercle. According to the general principle already enunciated, its efficacy is greatest in the strictly localised and least massive forms. But when we inquire how it acts, and what is the precise aim in its use, the difficulties begin to appear. As a rule, a rise in opsonic index is attended with improvement in the condition of the patient. As Latham has shown at the Brompton Hospital,(25) a skilful observer can, after sufficient experience in Wright's methods, by closely watching the clinical course of a case of phthisis under tuberculin, obtain data for his guidance closely corresponding with the movements of the opsonic index. This is a tribute to the general reliability of the index, as well as a relief from the necessity for its constant use. Sometimes, however, though the index is definitely raised, the patient is not better, or becomes worse, or, as Sir W. Watson Cheyne observed in tubercle of one epididymis, suffers from new infection on the other side, while the original lesion is healing.(26) Again, is not repair of tubercle due rather to tissue reaction than to phagocytosis? Are not polymorphonuclear leucocytes conspicuous by their absence in tubercle? Some observations on tubercle of the iris point decidedly to tissue reaction.(27) Do we aim at absorption or encapsulation? As a rule, absorption is limited, and encapsulation is the larger process, but it would seem that improved technique may cause wonderfully complete recovery. Do we range ourselves with those who strive to accustom the patient to larger and larger doses, carefully avoiding reaction, so as to establish an active immunity, or with those who would keep the tuberculous tissue hypersensitive by continued mild stimulation, and thus aid the textural processes which tend to resolution? Is Escherich right in his suggestion (28) that the immunising method is better for the adult, and the sensitising or so-called anaphylactic method better for children, especially in chronic cases with limited lesions and fair general conditions? As to dosage, do we take our stand with Schlossmann,(29) who says that antibodies (deviating complement) are not produced in children till doses of $\frac{1}{10}$ gramme are reached, and who commences in children with $\frac{1}{1000}$ to $\frac{1}{100}$ milligramme and increases rapidly to $\frac{1}{2}$ or 2 or even more grammes; or with those who, following Wright's more sensitive opsonic index, would commence with $\frac{1}{50000}$ milligramme and increase slowly to $\frac{1}{15000}$ or possibly to $\frac{1}{4000}$ milligramme as a maximum. British and American opinion, and I think I may add Australian opinion, on the whole inclines strongly to minimal doses.

Wright's researches into the complicated problems of auto-inoculation have been crowned by the remarkable results obtained by Dr. Marcus Paterson in the treatment of phthisis at the Frimley Sanatorium connected with Brompton Hospital. The patients are placed under ideal hygienic conditions with open-air treatment, and are kept at rest till fever is gone. Then auto-inoculation is induced by graduated exercise or work. Tuberculin is used to reinforce the treatment whenever necessary. If excessive auto-inoculation occurs, indicated by slight rise of temperature and headache, the patient is kept as rigidly at rest as if typhoid fever were present, till exercise can be resumed. The whole method is controlled by opsonic tests, which have been invaluable in revealing any faults of judgment in treatment. These wonderful observations have just been published in book form (30).

Sensibilised Vaccines.

The immunity gained by vaccination with anti-serum is usually of very short duration. That obtained by injecting cultures killed by heat is more durable, but the process may entail ill consequences both local and general. If anti-serum is added to cultures killed by heat, the dangers are removed, but the effects become transient. Besredka (31) has invented a new process, and claims that it gives very rapid immunity, of durable character, without risk either local or general, without any negative phase. Cultures are emulsified in normal saline, and mixed with anti-serum, unheated, strongly agglutinating. After twelve hours, the deposit of agglutinated bacteria is washed with normal saline till the last trace of serum is removed; and the soft paste remaining is emulsified with normal saline and sterilised by heat. The method has so far been used chiefly with laboratory animals, but in some diseases it has been applied to man with satisfactory results.

Serodiagnosis.

It is impossible in this address to deal with the enormous aid rendered in diagnosis by the study of antibodies, including agglutinins, precipitins, opsonins, bacteriolysins, hæmolysins, specific sensitisers such as the tuberculins, and complement deviators, as in the Bordet-Gengon and Wassermann reactions. I am permitted, however, to briefly direct your attention to the recent collective research that has been carried out in Melbourne concerning the diagnosis and prevalence of syphilis. The undertaking followed naturally upon the proceedings of the last Session of this Congress, which will be fresh in your memory. The Chairman of the Board of Public Health, Dr. Ham, induced the Government of Victoria to make provision for a collective investigation in the Metropolitan district of Melbourne during twelve months by means of the Wassermann test. An Advisory Committee was formed, consisting of Professor Allen (Chairman), Dr. Ham, Dr. J. W. Barrett, Dr. Hiller, Dr. Stawell, and Dr. A. Jeff. Wood, Dr. Ernest Jones being subsequently invited to join the Committee. Circulars were addressed to all medical practitioners in the metropolitan district, inviting them to forward a sample of blood from every case diagnosed as syphilis or open to suspicion of syphilis. A schedule was given not only of primary, secondary and tertiary syphilis, but also all cases of thoracic aneurism or aortic retroversion, all cases of apoplexy in young subjects, all case of locomotor ataxy and general paralysis of the insane, cases of congenital and infantile syphilis, all cases in which a mother gives birth to a syphilitic child, or has suffered from two abortions, or has lost three children from disease under five years of age, and any other suspicious cases. The reports were to be impersonal, and a fee was paid for each report. The conduct of the test was placed in the hands of Dr. Konrad Hiller, formerly Acting Director of the Bacteriological Laboratories in the University of Melbourne, and he performed all the tests. Five thousand distinct cases were reported. In addition, Dr. Barrett during four months submitted to Dr. Hiller's testing every case attending his clinique at the Eye and Ear Hospital as in-patient or out-patient for any cause whatever. Moreover, in 100 consecutive post-mortems made by him at the Children's Hospital, Dr. Gilbert Lamble, Stewart Lecturer and Demonstrator of Pathology in my department, conducted a microscopic examination of tissues alongside of the Wassermann test by Dr. Hiller.

Out of 5,000 cases reported (not including those of Dr. Barrett) 1,900 gave a positive reaction, and over 400 a partial reaction. The 5,000 cases included a large proportion of instances of tertiary and latent syphilis; and it is therefore in accord with large experience to assume that out of every 100 cases of syphilis, only about sixty would give a positive reaction, or, in other words, that every sixty positives would represent 100 cases of syphilis. Nineteen hundred positives would therefore represent 3,167 cases of syphilis, or 63·3 per cent. of the 5,000 reported.

In certain large practices, no reports were made. In many practices, large and small, it was only possible to report in a fraction, sometimes only a small fraction, of the possible cases. Hence the total amount of syphilis must be sought in other ways.

At this point Dr. Barrett's results become very significant. Out of 443 eye cases, of whom the great majority came to be fitted with spectacles. thirty-five gave a positive reaction, and twenty-three a partial reaction, The positive cases form 7·9 per cent.; the positives and partials together over 13 per cent. Out of 107 ear, throat, and nose cases, nine gave a positive reaction, and eight a partial reaction, the positives being 8·4 per cent., the positives and partials together 15·8 per cent. In totality, of 550 cases, 44 were positive or 8 per cent.; and, by the interpretation already given, this represents about 13·3 per cent. of syphilis in the entire clinique. The positives and partials, taken together, form 13·6 per cent. of the clinique.

Dr. Lamble's results show that there is the closest possible correspondence in children between the results of the Wassermann test and those of the histological tests which I described at the last Session of this Congress.

These findings are of great moment to the community, and I commend to the earnest consideration of the Congress the papers which will be submitted by Dr. Hiller and Dr. Lamble at the meeting concerning syphilis. The provisional estimate at which I have arrived is that probably about 7 per cent. of the population in the metropolitan district of Melbourne are infected in some stage or degree; and there is no reason to suppose that Melbourne is in worse plight than any other capital city.

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A CONTRIBUTION TO A DISCUSSION ON SERUM AND VACCINE THERAPY

At the Ninth Australasian Medical Congress, Sydney, September, 1911.

By D. A. Welsh, M.D., F.R.C.P. (Edin.).

Professor of Pathology, University of Sydney, and Honorary Pathologist, Royal Prince Alfred Hospital.

Professor Allen has asked me to reply on his behalf, and my undertaking is made easier by the fact that this great gathering has shown in no uncertain way that it recognises the high place which scientific immunisation has taken in the treatment of bacterial disease. If I now take exception to some points of detail, and even if I ask you to reconsider the statement of certain principles of treatment, I would not have you think that this represents any fundamental divergence. I go as far as those who have already spoken, but I think there are good reasons for venturing further than some of them have done.

Antisera and their Administration.

In regard to antisera, I would deprecate any suggestion that "mild" cases of diphtheria may safely be left untreated by antitoxin. No one can predict that a case will continue "mild" to the end, and the days of grace, within which antitoxin is effective, quickly pass. Careful observation and experiment teach that success in the use of antitoxic sera depends on the *early* introduction of *large* doses. To neglect the use of antitoxin even in "mild" diphtheria is no longer a legitimate form of research. It is not even original, for the disastrous results of such neglect were more than sufficiently illustrated in the pre-antitoxin days. Indeed, it is good practice to give a large dose of antitoxin as soon as there is reason to suspect diphtheria, without waiting for a report on the bacteria found. Much harm may thus be averted, if the suspicion should be confirmed; and no harm is done, if it should not.

A large and early dose of antitoxin is still more urgently indicated in tetanus, where not less than 100 c.c. should be given without the loss of a single hour, and followed by other injections. Here again it is good practice to anticipate possible developments by giving 25 c.c. or more of tetanus antitoxin, when wounds have been inflicted by rusty nails, or contaminated by earth, before there is any sign of tetanus, and so to place the injections as best to antagonise the local production of the toxin and its absorption by way of the nerves.

The Rationale of Vaccination.

It is in regard to vaccines that I am most interested, and in that modern development of vaccination which we owe to Wright. Vaccination, as I understand it, is the scientific direction of nature's mode of cure. It reproduces and regulates the mechanism by which recovery from infection is naturally brought about. The success of such a method is assured, and it is bound to succeed in ever greater measure as we reach a better understanding of its principles. What, then, is nature's method? It may be crudely stated as the formation of antidotes by the interaction between the infecting bacteria and the infected tissue. But most often the attack of the invading bacteria is concentrated in certain tissues; and, whether this concentration be limited to one area, or distributed over several areas, the result is the same—that each invaded area is liable to be overwhelmed by the numbers and virulence of the invaders, and its capacity for resistance to become exhausted. At the best this focal concentration of bacteria tends to derange the local mechanism of resistance, and to make the process of immunisation irregular, uncertain, and protracted. Hence it is necessary to make a counter attack—of the nature of a flank attack—and to bring into action relays of unexhausted tissue.

This is done by injecting a vaccine (usually a sterilised culture of the bacteria concerned) into an area of healthy tissue to provoke an immunising response there, so that the site of inoculation becomes a local manufactory of the anti-substances required. The bacteria are killed before being introduced, so that they cannot multiply and get out of hand; and the dose of bacteria is so regulated that, as Wright puts it, the tissues must win at the site of inoculation. The excess of anti-substances, thus generated in healthy tissue, is carried in the lymph and blood streams to antagonise the bacteria concerned in the infection, and so to reinforce the other tissues that are being overcome by them.

Certain principles of treatment obviously follow. It is inadvisable to exhaust the local manufactory of antidote by giving repeated injections of vaccine into the same area. The sites of vaccination should, if possible, be so chosen as to bring the anti-substances to bear upon the foci of infection in their most concentrated form. Autogenous vaccines should be used to develop the maximum antagonising power in the antidotes. Auto-inoculation should be carefully controlled during the period of immunisation. All other means that may accelerate the immunising response, or combat the infection, should be brought into action. Unkind criticism may then suggest that the patient has got well in spite of vaccination. But it must be admitted that the whole object of treatment in bacterial disease is the establishment of an immunity; and even those who believe that this is best done by allowing nature to work out a cure, should bear in mind that auto-vaccination is nature's mode of cure. It surely follows that this very method, scientifically directed, is the best means of treatment, and that all other means are subordinate and ancillary to it.

Vaccine treatment is therefore applicable to all bacterial infections—localised or generalised—acute or chronic—severe or slight. Acute local infections may respond to one or two injections. Chronic lesions may require prolonged and repeated vaccination. I was led by the work of Wright himself to believe that, even in the most acute and generalised conditions, a vaccine might help; that, indeed, a vaccine is the only thing likely to help. For one does not add to the *toxic* content of the blood by introducing a vaccine into the subcutaneous tissue. What one does is to stimulate the local production of anti-substances which, being absorbed, increase the *immune* content of the blood. In the most acute septicæmia, therefore, a vaccine should be promptly used, and in a number of hospital cases we have had a measure of success.

It is fortunate that the popular prejudice against vaccination for smallpox does not extend to vaccine therapy. Most patients are keenly interested in bacterial vaccines, and are more than willing to try them. I cannot help thinking that "conscientious" objectors would be less in evidence if the true nature of the process were known—if it were realised that vaccination is the adaptation of a natural process—the natural mode of recovery. Medical men should take every chance of impressing their patients with this aspect of the question.

Some Practical Applications and Suggestions for the Use of Vaccines.

I was impressed by the first case on which I tried a vaccine. It was that of a young woman with early phthisis and with laryngeal tuberculosis so advanced that a snipping from the mucous membrane revealed numerous giant-cell systems and tubercle bacilli. Minute doses of tuberculin T.R. were given, as recommended by Wright, and controlled by the opsonic index. Within a year every trace of phthisis had gone, and only some congestion of the mucosa was noticeable in the larynx. Treatment by tuberculin in small doses was continued at intervals for another year when recovery was complete. It is now more than seven years since I first saw her, and I heard only the other day that she was still in perfect health. In my limited experience of tuberculin I have continued to recommend minimal doses, and have seen further evidence of their curative value. Active processes have been arrested in the lungs, in glands, and in foci elsewhere, but masses of tuberculous tissue may not be absorbed, and the immunisation may have to be supplemented by operation. Advanced pulmonary lesions have not been much influenced. In genito-urinary tuberculosis there has been improvement even in advanced cases, and the subjects have repeatedly expressed a sense of well-being during these injections.

In non-tuberculous infections of the respiratory tract, I have known vaccines, prepared from bacteria in the sputum, give relief in asthma, cure a distressing condition of bronchiectasis, and abolish post-nasal and pharyngeal catarrh. Among other conditions I have seen recovery or marked improvement follow the use of autogenous vaccines in the cystitis of children and of adults, in extensive ulceration due to streptococci and otherwise intractable, in chronic rheumatism, and in suppurative processes, including carbuncles, boils, and acne. Failures I have known, and I am prepared to admit that most failures are evidence of faulty vaccination. I cannot agree that they discredit the principles on which vaccine therapy is based. I consider that there should be no failure in treating a bacterial infection with its autogenous vaccine save in two circumstances—(*a*) when the immunising response is defective, or (*b*) when the immune substances cannot reach the bacteria.

Two suggestions, arising out of work in this school, I would make for the further use of vaccines:—(1) in treating enteric fever and allied infections; and (2) in disinfecting the carriers of infection. Enteric fever begins as a septicæmia, and Dr. Hamilton Tebbutt,* in 1910, working as Resident Pathologist at the Royal Prince Alfred Hospital, found that in early conditions he could make the diagnosis from cultures of the bacilli in the blood, more certainly than by testing for specific agglutinins in the serum. Autogenous vaccines from such cultures could be used at an early stage of the infection, before the bacilli had had time to settle and damage the tissues. A beginning has been made by Dr. Tebbutt and by Dr. Mary Burfitt at the hospital with encouraging results. If it should prove helpful to use vaccines in enteric fever, it should be helpful to use them in other infections which concentrate in the alimentary tract. In the gastro-enteritis of children, Dr. Jean Dalyell is making a special study of the bacteria concerned, with the object of applying specific vaccine therapy. In dysentery and in outbreaks of food poisoning the use of vaccines is also worthy of trial.

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* A report on the bacteriology of typhoid fever, *Austral. Med. Gazette*, 20th April, 1910.

That vaccines might be used to disinfect the carriers of infection was brought under my notice some years ago by Dr. Percy Walton Smith, who used small doses of a recent culture from a case in which the bacilli persisted in the fauces after recovery from diphtheria. This persistence of diphtheria bacilli during and after convalescence is by no means uncommon. It is quite unsuspected either by the patient or by his medical adviser unless a special bacteriological examination is made. It is a menace to the community against which no precaution is taken except by a few more enlightened members of the profession. In all probability it is the result—and the only bad result—of the use of antitoxin, which may neutralise the poisons of the bacilli before the tissues elaborate enough anti-bacterial substances to destroy the bacilli themselves. The use of a vaccine to kill off the bacilli is necessary in such cases, and it would probably be better for all diphtheria patients if the vaccine were administered as regularly and at as early a stage as the antitoxin.

Since a vaccine is effective in clearing the throat of diphtheria carriers, a corresponding vaccine might be able to clear the intestine of typhoid carriers. It is true that the typhoid bacilli in such cases might not be easily reached by the anti-substances produced by vaccination. But, when immunisation is sufficiently advanced, the anti-bacterial substances in the blood might be brought into contact with the bacilli by means of a saline purge.

Vaccines in General Hospital Practice.

In conclusion, I would bring under your notice the vaccine work of the Royal Prince Alfred Hospital as evidence of the appreciation of vaccine therapy by the Honorary Staff. All the vaccines are prepared at the request of the staff, and I am indebted for the following particulars to Dr. Mary Burfitt, the Resident Pathologist, who made the vaccines and helped in their administration. Within a period of eight months, February to September, 1911, autogenous vaccines were prepared from and administered to, 100 patients, and occasionally more than one vaccine was made from the same case. Since the daily average number of in-patients for that period was 357, and the weekly average of out-patients was about 200, the proportion of cases receiving treatment by vaccines is higher than it was in some of the British hospitals which I recently visited.

Details of a consecutive series of 100 unselected cases are given in the accompanying table. These results, however, do not give a just estimate of the efficacy of vaccines in the treatment of bacterial infections; for some patients did not come under treatment until late in the disease; others have only recently begun vaccination; and many did not attend long enough to complete the process of immunisation, otherwise more would probably have been "cured." Per contra, many patients would doubtless have been relieved without the aid of vaccines, though probably not within the same time, nor to the same extent. My object is rather to give some idea of the extent to which vaccines are actually being used in one particular hospital, now that the methods introduced by Wright have been put to the test for several years.

My own conclusion, based on these and on many earlier results, is that vaccines are of great value in the treatment of hospital cases, because some lives are saved in acute cases that would, in all probability, otherwise have been lost, and because many disabling infections are more quickly arrested, and more often cured, with vaccines than without. It is not often indeed that one can so conclusively justify to the lay mind the expense of a well-equipped pathological laboratory; but even the least sympathetic can understand that money is well spent when it enables a hospital to treat more patients in the same time and to treat them more effectively.

TABLE giving details of 100 consecutive cases treated by Autogenous Vaccines at R.P.A. Hospital within a period of eight months.

Nature of Cases.	Number of Cases and Results.				Remarks.
	Cured.	Improved.	Unimproved.	Died.	
Enteric fever	17	...	...	1*	*Admitted in 5th week, and died of hæmorrhage after one dose of vaccine.
Paratyphoid fever ...	2	...	...	...	
Diphtheria carrier ...	1	...	...	...	From a case of "sore throat" in a member of the Resident Staff.
Septicæmia (streptococcal)	3	1†	...	7	†Under treatment.
Septic endocarditis ...	...	...	...	2	Both very late in coming under treatment.
Osteomyelitis	...	1	1	...	
Pneumonia	1	...	...	...	
Pneumococcal empyema ...	1	2‡	...	...	‡Under treatment.
Pulmonary tuberculosis ...	...	1	...	...	Pneumobacillis isolated.
Abscess of lung	...	...	1	...	Improved, then relapsed, and a piece of hydatid membrane was coughed up.
Abscess of breast	1	1	...	...	Both multiple abscesses.
Pustular conditions of skin	5	15	2	...	More than one vaccine used in some cases.
Septic wounds, ulcers, &c.	...	5	1	...	
Cystitis and pyelitis ...	4	15	3§	...	§Under treatment.
Bacilluria	...	1	...	...	
Chronic (rheumatic ?) arthritis.	1*	3	1	...	*Streptococci and staphylococci both isolated; of the remainder two were streptococcal, and two staphylococcal.
Totals	36	45	9	10	

DISCUSSION ON SERUM AND VACCINE THERAPY.

By W. F. Litchfield, M.B., (Syd.)

I would like to say a few words on the serum treatment of diphtheria. Although the great value of this treatment is universally recognised, there are some phases of it on which views are by no means uniform. I am thinking especially of the dosage, the value of diphtheria antitoxin by the mouth, and the phenomena due to anaphylaxis, or, in other words, the exaggerated serum-sickness that occurs when a second injection is given fifteen days or more after the first one. In 1907 Dr. Clubbe reported the comparative results of the last 300 cases treated without antitoxin and the first 300 cases treated with antitoxin at the Children's Hospital, Sydney. They were as follows:—

Last 300 cases without antitoxin—total mortality 52 per cent. Of these, 199, or 66·2 per cent., were tracheotomy cases, of whom 67·8 per cent. died.

First 300 cases with antitoxin—total mortality 20 per cent. Of these, 129, or 43 per cent., were tracheotomy cases, of whom 37·9 per cent. died.

The doses of antitoxin given in these cases ranged from 500 units to 4,000 units, and the result was a great reduction in the total mortality, the mortality in tracheotomy cases, and in the proportion of tracheotomy cases to the whole number.

To compare with these results I have taken the cases treated at the same hospital for 1909-1910 and the first half of 1911. There were 635 cases, with a total mortality of 7·4 per cent. Of these, 184, or 28·9 per cent., needed operation; and of the 184 operation cases twenty-four, or 13 per cent., died.

In these cases much larger doses of antitoxin were employed, namely, from 4,000 to 30,000 units for pharynxeal diphtheria; 15,000 to 30,000 units for larynxeal diphtheria; and from 50,000 to 150,000 for malignant diphtheria. In the three series the total mortalities were 52 per cent., 20 per cent., and 7·4 per cent.; the proportions of operation cases to the total, 66·2 per cent., 43 per cent., and 28·9 per cent.; and the mortality of operation cases 67·8 per cent., 37·9 per cent., and 13 per cent. The cases treated at the Children's Hospital are of high average severity, owing to the number of beds being limited, and the results given include those dying within a few hours of admission. The above results show that for the effective treatment of diphtheria large doses of antitoxin are required. No precise rules can be laid down as to dosage, but it is better to give more than necessary than to give too little. Our experience, too, is that it is better to give the whole dose at once rather than to divide it. The antitoxin is given subcutaneously, except in very urgent cases, when portion of it is given intravenously. The largest dose given by us was 170,000 units, 50,000 of which were given intravenously. The case was one of malignant diphtheria in a boy aged 5 years, and he recovered. Of antitoxin given by the mouth we have had practically no experience, and are not in a position to approve of or condemn it. Many practitioners use diphtheria antitoxin by the mouth, and the method is such a convenient one that it is a pity that some authoritative statement on the subject is not available.

With regard to the serum reaction following a second injection after more than fifteen days, or anaphylaxis as it is called, we have not kept systematic records; but we can say, though there have been a good many cases receiving two such injections, no deaths have occurred from the reaction. One child, aged 2 years and 5 months, had a second injection of 20,000 units after an injection of 10,000 units twelve months previously. He developed severe urticaria on the second day, had another crop on the fourth day, and another on the ninth day after the second injection. Another child, aged 2 years, had a second injection of 4,000 units after a first injection of 2,000 units twelve months previously, but up to the ninth day did not develop any urticaria. One of our nurses had very severe urticaria and pains in the joints after a second injection, the doses being 6,000 and 12,000 units, and the interval thirteen months. One of our house surgeons had a similar experience. In the case of a nurse who had, after a previous injection, rather severe urticaria, and who was subject to asthmatical attacks, I adopted the method of giving a second dose, rendered necessary by an attack of pharynxeal diphtheria, by the mouth. No urticaria developed, and the throat became clear of membrane in two days.

I would also like to refer to two cases of cerebro-spinal meningitis that I treated with Flexner's antimeningo-coccal serum. The first was a half-caste coloured girl, aged 7 years. She was admitted to the Children's Hospital in February, 1910, and was said to have taken ill the previous day. She had high fever, delirium, stiffness of the neck, and a widespread crop of purpurous hæmorrhages. Lumbar puncture was done, and about two test tubes full of turbid fluid, containing pus cells and intracellular gram negative diplococci. The contents of one bottle of Flexner's serum was injected into the spinal canal immediately after the lumbar puncture. These proceedings were repeated on the next three mornings, the child's condition becoming less acute each day. At the end of the first week it was noticed that she was deaf. She had a prolonged convalescence, but eventually made a complete recovery except that she was left quite deaf. The other case was a boy, aged 3 years, whose symptoms were far less acute. He had three injections at intervals of a few days, and after a prolonged convalescence also recovered, but, like the other patient, was left quite deaf. It is not possible to speak with certainty about these cases, but I think the serum was responsible for the first patient's recovery.

INFANTILE PARALYSIS.

PAPERS AND DISCUSSION IN THE SECTIONS OF DISEASES OF CHILDREN, NEUROLOGY, PATHOLOGY, AND PUBLIC HEALTH.

Thursday, 27th September, 1911.

NOTES ON THE PATHOLOGY AND ÆTIOLOGY OF ACUTE ANTERIOR POLIOMYELITIS.

By Wilton W. Love, M.B., C.M. (Edin.).

In my college days, a quarter of a century ago, we were taught that the pathological lesions to be found in cases dead of acute anterior poliomyelitis, consisted of atrophy of the ganglion cells in the grey matter of the anterior cornua of the cord, as demonstrated by Charcot; but as to the cause of this atrophy both professors and books were alike silent. The muscular atrophy caused by the destruction of the ganglion cells was due to trophic changes and fatty degeneration.

Isolated cases in the chronic stage occasionally cropped up, during the years of my service at the Children's Hospital in Brisbane, but threw no fresh light on their ætiology. About 1896 I received a letter from Professor Starr, of Philadelphia, asking if any epidemic prevalence of the disease had been observed in Brisbane, which I was obliged to answer in the negative, and at the same time I expressed surprise as to any epidemic character of the malady This doubt as to its epidemicity was dispelled by the outbreak of 1905, when a considerable number of cases was observed in Brisbane. The pathology of the disease had made no advance since the observations of Charcot and Roger in 1872, and we were still in the dark as to causation and mode of transference. Two years ago this month, the veil was partly lifted by the important researches of Landsteiner and Popper, and later of Flexner and Lewis, of the Rockfeller Institute, in their reports on "Experimental Poliomyelitis in Monkeys." Up to this date the only definite knowledge we had was the morbid histology of the cord and the muscles and nerves of the affected parts. No causal micro-organism had been isolated, and no experimental reproduction of the disease had, so far, been achieved. In 1909, however, Landsteiner and Popper had succeeded in transmitting the disease to monkeys by the injection of spinal cord emulsion from a child dead of acute poliomyelitis—following the method of Pasteur in his rabies experiments; but they were unable to reproduce the disease by further inoculation from these monkeys. It remained for Flexner and Lewis, of New York, to succeed in transmitting the disease to a successive series of monkeys. In the absence of any discoverable germ, Pasteur's method of using the spinal marrow itself for inoculation was used.

The cord was emulsified in salt solution and injected into the substance of the brain or into the peritoneal cavity. After an incubation period of about ten days on an average, clinical phenomena corresponding in all respects to those of acute anterior poliomyelitis were observed, the paralysis involving some or all of the limbs, and terminating after a variable period in recovery or death. The mortality in monkeys proved to be much higher than in man, reaching 50 per cent. or slightly over.

Landsteinet and Popper found experimentally by the peritoneal route:

(1) Internal organs normal.

(2) Spinal cord showed severe lesions.

Pia mater infiltrated with small, round, deep-staining cells, the infiltration extending along anterior median fissure and less along posterior median fissure into cord, medulla, and brain. Areas of inflammation, especially at cervical and lumbar enlargements, showing perivascular infiltration or diffuse infiltration into substance of cord. Œdema of meninges noted. Diffuse infiltration, chiefly in anterior grey matter, and to a less extent in posterior grey matter. Similar lesions, but less intense, were noted in pons medulla and crura cerebri. Severe degeneration of ganglion cells in anterior horns, the ganglion cells being surrounded, or even invaded, by cells. Hæmorrhages into grey matter.

Flexner and Lewis found, by intra-cerebral injection: Cord and membranes congested, especially at cervical and lumbar enlargements; no increase of cerebro-spinal fluid; consistence of cord normal; punctiform hæmorrhages; cord more affected than brain; no part of cord escapes altogether; meninges infiltrated with perivascular infiltration, causing compression of smaller vessels; grey cornua most affected; cellular infiltration always interstitial, never exudative on surface; chiefly lymphocytes, with very few polynuclears. Infiltration extends along nerves, especially by posterior roots; white matter infiltrated, showing occasional hæmorrhages. Grey matter more affected than white, especially in anterior horns; medulla and brain showed similar, but less intense, lesions; intervertebral ganglia affected.

The observations of both observers, in each case, show a perfect reproduction of the lesions described by Charcot, Roger, and Damaschino.

Hektoen, in his "Pathology," affirms that the pathogenic agent acts primarily on the blood vessels, and secondarily on the ganglion cells, the histological appearance being identical with those produced in the experimental cases.

To sum up the pathological conclusions derived from previous observations, and from recent experimental work, we find (Osler) that:

(1) The disease is faultily named Infantile Paralysis, as it is found in adults as well as in children and infants.

(2) The morbid anatomy of the epidemic cases is similar to that of the sporadic cases.

(3) The changes in the adult are the same as in the child.

(4) The medulla and brain-stem, and even cerebellum and cerebrum, are also affected in severe cases.

(5) The extent of the lesions is usually found to be far greater than the clinical symptoms would suggest.

(6) The morbid histology shows acute congestion of meninges and cord; congestion of all blood-vessels in white and grey matter, and packing of their adventitial sheaths and lymph channels with cells; an intense proliferation or exudation of cells (concerning the origin of many of which there is a difference of opinion), limited almost entirely to those parts of the central nervous system most freely supplied with blood, viz., the neighbourhood of the rich capillary network of the grey matter of the cord, and to the well-vascularised regions of the brain-stem and brain, with subsequent retrograde changes in those nerve-cells and nerve-fibres which are intimately involved in the areas of inflammation.

The bacteriology of the disease is still unsettled. Flexner and Lewis found no organisms stainable in smears or sections from affected cords, but found that the virus could be filtered through a bacteria-tight filter, these filtrates regularly causing paralysis in inoculated monkeys. The clear filtrates, when examined under dark-ground illumination, showed innumerable bright dancing spots, devoid of definite size and form, and not truly mobile.

Stained with Loeffler's flagella-stain, this filtrate shows minute particles of round or oval form, which were absent from a similar filtrate prepared with the nervous system of a rabbit. These appearances are also observed in subcultures in bouillon prepared with blood serum or ascitic fluid; but attempts to induce paralysis by injections of subcultures have so far been unsuccessful.

The virulence of the filtrate is easily destroyed by heat, but does not seem to be easily impaired by cold.

Ætiology and Distribution.—The epidemic character of acute anterior poliomyelitis has been amply proved of recent years by the epidemics which have visited the United States in 1907, and Continental Europe in 1909, and Australia in 1905 and the summer of 1907. No direct evidence of infectiousness or contagiousness has yet been forthcoming; as Flexner found that healthy monkeys could be kept in the same cage as infected ones without contracting the disease.

With regard to the avenues and sources of infection, Flexner found that the disease could be inoculated by the intra-cerebral or intra-peritoneal routes, through the blood, cutis, spinal canal, and large nerves, but not by feeding or intra-tracheal injections. He found that the naso-pharyngeal mucosa contained the virus, as macerations of the mucous membranes, tonsils, and turbinated mucosa with quartz sand caused paralysis on being injected into the brain. Landsteiner and Levaditi found salivary glands sometimes infective.

Some attacks in monkeys proved abortive, and these monkeys were insusceptible to further inoculation. Flexner and Lewis were unable to infect guinea-pigs, rabbits, horses, calves, goats, pigs, sheep, dogs, or cats.

Experiments by Cushing and Crowe show that flies which were allowed to feed on infected cords were capable of producing paralysis on injection intra-cerebrally after filtration of the macerated bodies.

Flexner concludes his report by stating that the experimental study of poliomyelitis has yielded a large number of important facts relating to the spontaneous disease in man. The nature of the virus has been discovered, some of its immunity effects have been established, the clinical and pathological peculiarities of the disease have been elucidated, and a basis has been secured on which to develop measures of prevention.

DIAGNOSIS AND SYMPTOMATOLOGY OF INFANTILE PARALYSIS.

By A. Jeffreys Wood, M.D. (Melb.).

The diagnosis of acute anterior poliomyelitis has, in the light of Flexner's recent experiments on monkeys, become of the greatest moment. He has shown that where he has treated monkeys with urotropin before inoculating them with the poison of the disease, it is possible to prevent the development of the disease, whilst the control monkeys developed it. If then it were possible to diagnose the probable onset of anterior poliomyelitis in its preliminary stage before the onset of paralysis, we might modify, if not prevent, the subsequent paralysis.

During an epidemic of the disease those who work amongst children naturally look with the gravest suspicion on every slight feverish attack, attended with vomiting and disturbed sleep; but up to the present time our hands have been tied, and no symptoms have revealed themselves that might be looked upon as being in the least pathognomonic of the disease. Recent experimental work, however, shows that there is a distinct change in the character of the cerebro-spinal fluid during the preliminary stage of the disease.

The cerebro-spinal fluid from a healthy child shows that it is rare to find more than about three cells to the cubic millimetre. Flexner has shown that in monkeys, twenty-four hours after inoculation, the cerebro-spinal fluid was normal in quantity, but the small cells had increased in number; this increase is more marked in forty-eight hours.

Lucas showed that in monkeys, after inoculation, the cells increased from the normal three cells to 100 or 300 cells per cubic millimetre during the incubation period. Lucas also examined the cerebro-spinal fluid from four patients suffering from anterior poliomyelitis whom he saw on the second to the fifth day of the disease, when they were still hypersensitive, and paralysis was just appearing. The number of cells in the fluid at the first puncture varied between 50 and 350. This increase in the number of cells was maintained in all the cases up to the last examinations, the latest of which was thirteen days after the first onset of symptoms. The lymphocytes and small mononuclears predominated at the first examinations. Subsequently large mononuclears appeared, and finally polynuclears began to appear. He found that the results with regard to the type of cells agreed with his findings in the cerebro-spinal fluid of inoculated monkeys. The character of the cerebro-spinal fluid, then in the acute stage of poliomyelitis, is as follows:—It is clear and not infrequently under somewhat increased pressure. It sometimes shows a fibrin clot on standing. It always contains an excess of cells, these cells being chiefly of the mononuclear type, most of them being lymphocytes, and above all these changes are present before the appearance of the paralysis. Unfortunately, however, these are the characteristics of the cerebro-spinal fluid from cases of tubercular meningitis. It is, of course, true that tubercle bacilli are present in this latter fluid, but our experience shows that they are not always found. The cases of tubercular meningitis are usually more slow in their development than cases of anterior poliomyelitis, so that during an epidemic of infantile paralysis a cell count of the cerebro-spinal fluid of a child taken suddenly ill would be a good indication whether to make a diagnosis of poliomyelitis or not, and so treat the case effectively.

Blood-counts in the case of monkeys were done by Lucas, who showed that there was a marked drop in the number of white cells during the irritative or prodromal stage, but this leucopenia, which occurs in animals, does not apparently occur in man, for the results obtained by both Lucas and Morse show that there is frequently a leucocytosis. It remains for future observers to say whether there is a lymphocytosis or not in the early stage in man; there is in monkeys. If we except the change in the character of the cerebro-spinal fluid in the prodromal malaise of infantile paralysis, there is absolutely no indication amongst the symptoms to indicate the onset of this heart-breaking disease, which may cripple the child for life.

When the paralysis occurs there is little difficulty in making the diagnosis. In small infants syphilitic pseudo-paralysis, depending on acute epiphysitis, must be remembered, and in children over six months the apparent paralysis of one or more limbs occurring in cases of scurvy can, as a rule, be immediately detected by examining the gums for hæmorrhages.

The acute pain in the limbs and back, that forms such a prominent feature of the disease in the severe cases—50 to 58 per cent. of cases during an epidemic—is a symptom that may lead to a diagnosis of multiple neuritis, or even acute rheumatism. But the acute onset of the paralysis, in anterior poliomyelitis, the rapid wasting of the muscles, the spontaneous improvement in certain groups of muscles, the absence of sensory symptoms, and finally the reaction of degeneration in some of the muscles, all combine to form a type which it is difficult to confound with any other disease.

Symptoms of Infantile Paralysis.

In dealing with the symptoms of this disease, as revealed during the epidemic that occurred in Melbourne during the autumn and winter of 1908, a far broader view of the ravages of this disease in the cerebro-spinal system was found necessary than had previously been appreciated by the medical profession. As a matter of fact, all the types of the disease so admirably tabulated by Wickman were seen in our epidemic. His table of clinical types is as follows:—

1. Spinal poliomyelitis, which is by far the largest class.
2. Ascending myelitis, where the paralysis gradually ascends until even the basal nuclei are attacked. These cases are usually called Landry's paralysis, but they are recorded in the course of all epidemics, and undoubtedly are due to the same cause as the spinal poliomyelitis cases.
3. Bulbar or pontine cases, which are generally soon fatal, although one of my cases, after threatening to die of suffocation for two days, eventually made a perfect recovery.
4. Encephalitis: In these cases the cerebral cortex is attacked, convulsions are usually marked, and spastic paraplegia, and possibly idiocy, are the results in many cases.
5. Ataxia in cases where the disease affects the cerebellum. In these cases nystagmus, scanning speech, and hypotonus of the limbs may be added to the ataxic symptoms.
6. Polyneuritis.
7. Meningitis, when symptoms of meningeal inflammation develop before the paralysis.
8. Abortive forms occurring in families alongside other definite cases, although showing no paralysis in the end. Anderson and Frost have recently shown that the blood from 66·7 of their abortive cases showed that it possessed the same power to neutralise the virus of the disease that the blood from true cases of poliomyelitis possesses.

General Constitutional Symptoms.

As a rule, there are marked constitutional symptoms preceding the onset of paralysis in the majority of cases. In a few cases the paralysis occurs suddenly, without any history of previous symptoms of a constitutional nature, just as in every epidemic there are recorded abortive cases where the constitutional symptoms have been present, but no paralysis has developed.

The constitutional symptoms seen in the stage of invasion are, as a rule: Fever, ranging from 100 degrees F. to 102 degrees F., for about three days, disappearing, as a rule, before the paralysis occurs; tossing about and twitching in sleep, drowsiness and irritability when awake, and refusal of food. Vomiting is a frequent initial symptom; it may only occur once or twice, but in one of my cases from Tasmania the vomiting continued at intervals for forty-eight hours. Catarrhal symptoms are sometimes present, with running at the nose and slight cough. The tongue is often thickly coated and the bowels constipated. During the Melbourne epidemic, constipation was a marked feature in some cases, even before the onset of febrile symptoms, and in some cases it persisted for months after the onset of paralysis; this symptom was apparently the result of paralysis of the muscular power of the bowel. One feature that marked our epidemic was the frequency of retention of urine; in some cases the catheter had to be passed for as long as a week before the vesical centres recovered their tone. These symptoms, as a rule, occurred suddenly in cases of children who had previously enjoyed perfect health.

In a few cases the onset of the disease closely resembles tubercular meningitis, as in the following case reported by Dr. Morse, of Boston:—E.D., female, æt. 2½ years, well up to 11th October, 1910, when she vomited and had a convulsion. Twitching of extremities followed the convulsion for eight hours. This was most marked in the right hand. The twitching recurred intermittently until 15th October. She remained unconscious for five days. On the morning of the fifth day paralysis occurred in the left arm, and soon extended to the legs, after which time the diagnosis of anterior poliomyelitis was easy. On the day before the paralytic symptoms occurred a lumbar puncture was done; 35 c.c. of clear fluid was drawn off. It contained thirty-five cells to the cubic millimetre, all of which were lymphocytes; no tubercle bacilli were found, and cultures were sterile.

A Von Pirquet tuberculin skin test was positive.

The constitutional symptoms are in no way characteristic, but it is of extreme interest to record them accurately when occasion affords the opportunity, for we thereby see how the statement in text-books arose that this disease followed as a sequel to so many different diseases.

The onset of the local paralysis is sudden. The mother of a child 12 years old, who came from Urana, gave me the following history:—Her daughter was well up to a Saturday in December, 1910, when she became feverish, and had severe headache; she was better on the Sunday and Monday following, but on the Tuesday again had severe pain in the head and back of neck. These symptoms increased in severity on the Wednesday, and she was very restless all night, and feverish. On Thursday morning, or the sixth day from the onset of the first headache, she climbed up into a buggy to drive in to see Dr. McKnight, but at the end of the drive two hours later she had to be lifted out of the trap, as her right leg was paralysed. Some hours later she lost power in her left leg.

Another child, from Maffra, moved her legs well when put into a buggy, but whilst driving 12 miles she lost the power of lifting her right leg.

Other children are attacked when out for a walk, and have to be carried home.

The symptom that causes the greatest distress to the patient is the acute pain that accompanies many of the severer cases. In one case I saw with Dr. Allen, of Elsternwick, the child's shrill cries of pain were heard across the road. The duration of the acute pain varied a great deal, but in some cases lasted for twelve weeks or more. The slightest movement in these cases was accompanied by acute exacerbations of the pain. One mother told me that she had to bend over her infant to give her the breast, not daring to move the child even to change the napkin for three days.

The paralysis is associated at an early date with atrophy, with changes in the electrical reactions and a loss of reflex activity in the paralysed parts. The paralysed limbs are usually much colder than those not affected. A bad sign is when the paralysed limb wastes rapidly, and shows marked coldness. In a large number of cases the paralysis is somewhat widespread at the commencement, and spontaneous improvement follows with the clearing up of the œdema of the cord, leaving certain muscles seriously affected, which form the residue of the paralysis. These muscles may remain permanently damaged throughout the patient's life.

The residual paralysis is usually seen in the lower extremity, confined to a certain group of muscles, often leading to Talipes valgus or varus. The subsequent growth of the limb is frequently affected, and the consequent shortened limb may lead to the development of a lateral curve of the spine.

Paralysis of the trunk muscles of the back are occasionally affected, and are responsible for many serious cases of scoliosis, with marked rotation of the bodies of the vertebrœ.

SURGICAL TREATMENT OF INFANTILE PARALYSIS.

By W. Kent Hughes, M.D., C.M. (Melb.).

The treatment of infantile paralysis is divisible into two groups—

(1) That directed towards prevention of deformity;
(2) That directed towards the relief of deformity.

The first group includes the treatment during the acute and subacute stage, where our efforts should be directed towards helping the patient to recover in much the same way as we help him during any acute illness, the only difference being that in addition we must take care to support the muscles that are most affected.

It is a common error to suppose that only one or two muscles in a limb are affected. Nearly every muscle in a limb is more or less paralysed, and we must place the limb, if possible, in such a position that none are overstretched.

When the disease has passed into the chronic stage we must still prevent the weakened muscles from being overstretched, and, at the same time, by judicious active and passive movements, coax them into activity and well-being as far as possible, always remembering that a complete cure in a severe case of anterior poliomyelitis is an impossibility.

We can imagine that cases may occur so slight that complete recovery takes place in every muscle—just as the majority of the muscles recover in every case; but it is hard to see how such cases come under observation. Personally, I am convinced that complete recovery is a grotesque delusion.

In the early stages the patient belongs rather to the physician, though, unfortunately, he is too often left entirely to our learned, though seldom practical, colleague, and in consequence deformities are allowed to ensue.

It is safe to say that no deformity should ever arise in a case that was intelligently treated from the outset; but, of course, that does not mean that some paralysis will not remain.

No elaborate or complicated machinery is necessary for this. The lower and upper limb type fall into two main groups—those affecting severely the proximal and those the distal end of the limb. This simplifies your task in the early stages, for all you have to remember is, " Assist the weaker brother, and do not rob Peter to pay Paul."

This is easily accomplished by simple splints that support the weight of the limb and counteract the action of gravity.

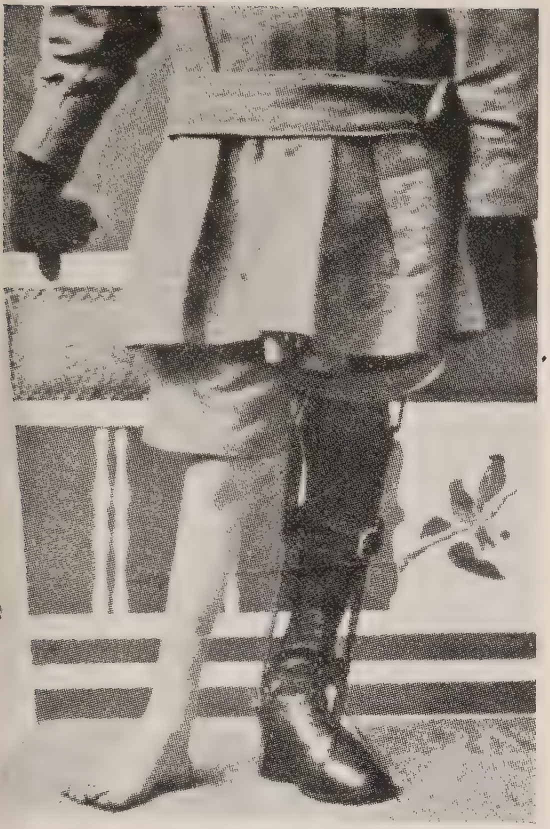
Second Group.—When deformity has occurred, which is the stage at which most orthopœdists see their patients, our efforts are directed to restore the limb to its natural shape and give the overstretched and weakened muscles a better chance to recover—and that as quickly as possible.

No one in his senses, and who wishes to do the best for his patient, woud ever dream of subjecting a patient to two years' splinting, and constant visitation by the surgeon, when two minutes cutting would effect the same end. At the same time, every encouragement, especially in wrist-drop, should be given to the overstretched muscle to recover before operation.

Amongst orthopœdist surgeons, the general practice is to procure lengthening of the contracted muscles by division, though I find that some excellent general surgeons who practice orthopœdy, pin their faith to splitting and lengthening, for fear, they say, of a weak union. I have never yet seen such a weak union; and they certainly make sure of portions of their lengthened tendon never being more than half the thickness of a normal tendon, for I find, curiously enough, that for some reason or other the gaps between a " Z " never fill up; perhaps the suturing has something to do with it. Another strong objection is that the usual incision is made directly over this tendon. If I cannot persuade them to abandon this method of lengthening, I hope they will incise the skin to one or other side of the tendon.

In all instances, except where there is danger—such as with the biceps tendon and popliteal nerve—I prefer subcutaneous tenotomy, as it is quicker to do and quicker to heal, and if the limb is to be put in a plaster, a skin incision is a decided disadvantage.

A point of difference, despite Syme of Edinburgh's teaching, is that some orthopœdists put the foot up in the deformed position after incision; the restoration is tedious and not so efficient, and does sometimes result in a weakened tendon.



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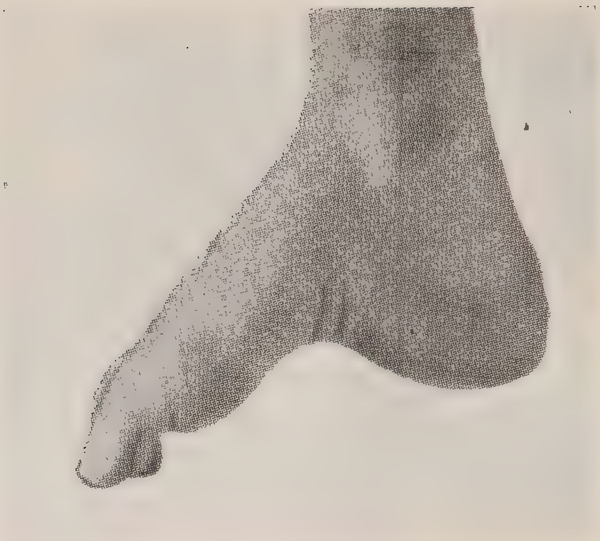

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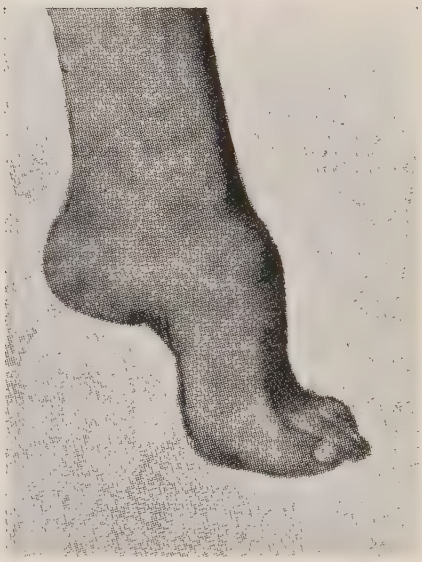

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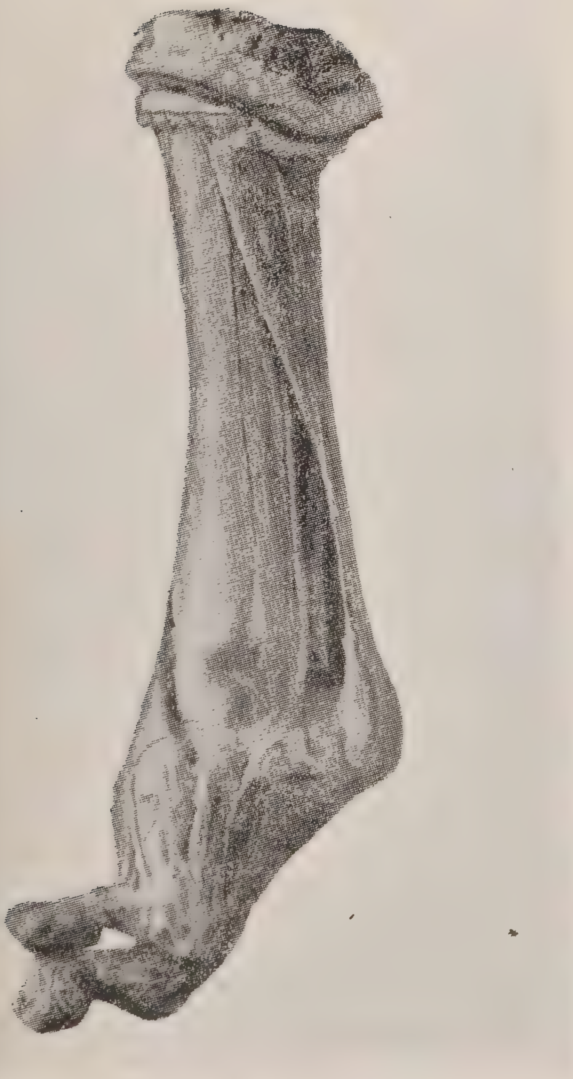

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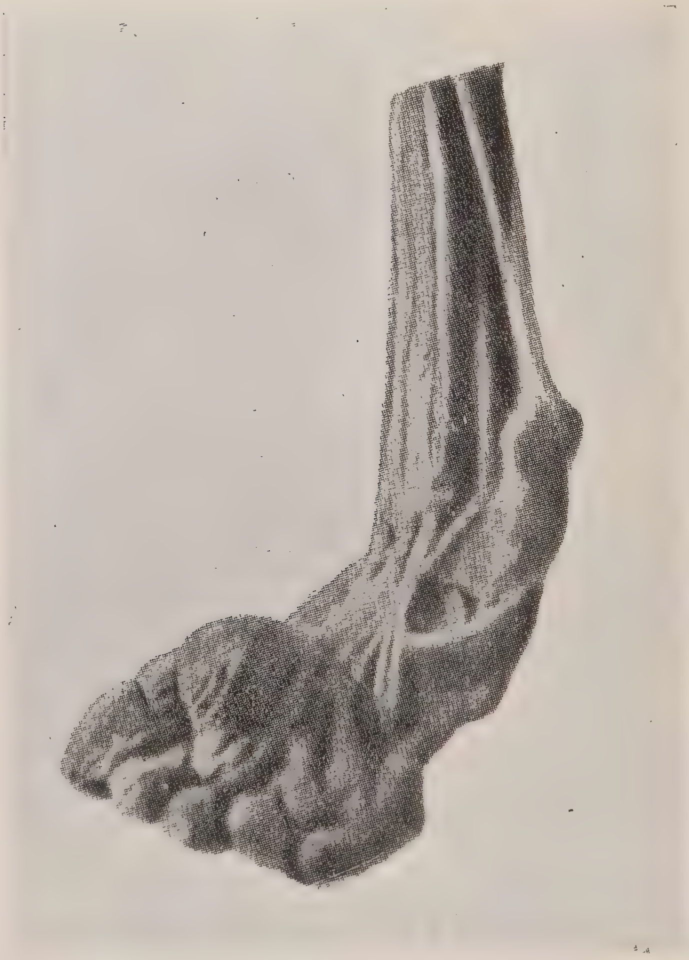

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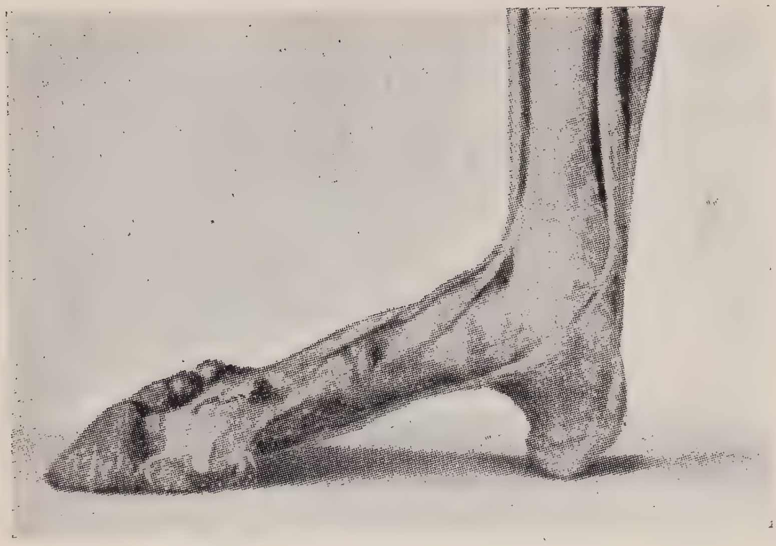

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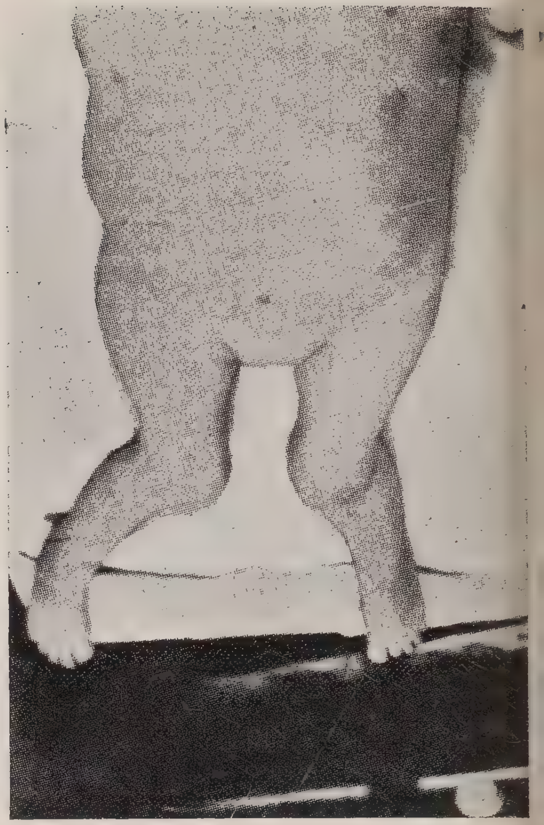

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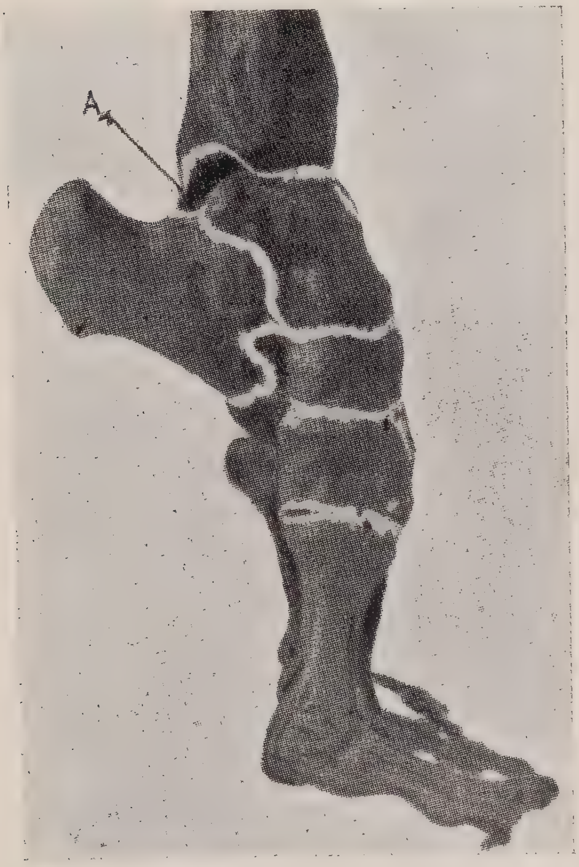

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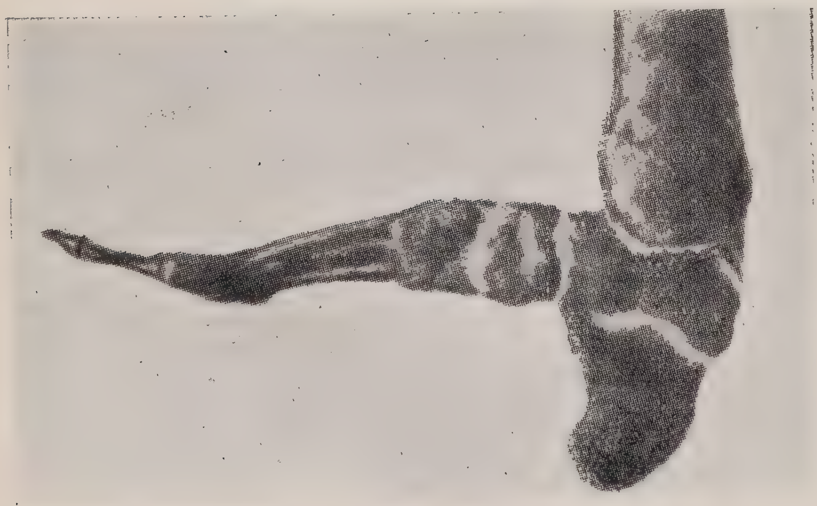

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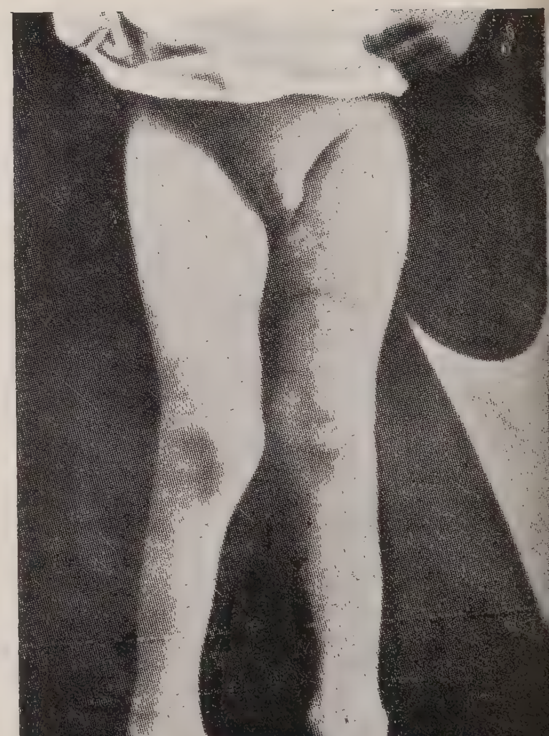

10.

Our aim is two-fold—(1) to abolish the deformity; (2) to help the weakened muscle. This can be most quickly accomplished by division and immediate rectification, and even if an occasional weakened union were the result, I should still persevere; but it has been my universal experience that perfect union always ensues, even in division and rectification of contracted hamstrings, and I have had separation of 3⅞ inches in tendo Achillis in a boy of 8, from Ipswich.

In dividing a tendon, we should always strive to maintain the integrity of the sheath as far as possible, even if only one layer can be kept intact.

In such a tendon as that of tendo Achillis, it is fairly easy to accomplish this by passing a sharp-pointed tenotome between the muscles and its sheath. The sense of touch will determine whether you have hit off the right position; then pass your blunt-pointed tenotome along the blade of the other and cut cautiously towards the surface while the tendon is stretched.

Where a wide gap is necessary, it is of course not possible to preserve the sheath entirely, but it is seldom that a complete rupture is necessary; if it is thought that rupture is probable, the division of the tendon should take place as near its attachment to the os calcis as possible.

After division of tendon the foot is put up in plaster for three days, and then the patient is encouraged to walk about with more or less support. In quite young children, none is required. In all tenotomies early movement is absolutely essential for perfect success.

To pass from general to special detail, might I direct your attention to illustration No. 3. You will see there a case of Talipes equinus, and you will observe that the deformity is primarily due to a drop of the anterior part of the foot at the medio tarsal joint and only secondarily to the contraction of the tendo Achillis—that is to say, it is due rather to the weakening of certain muscles than to the contraction of certain others. If it were not so, the position of the os calcis would be almost vertical, as in No. 1, which exhibits a more rare condition, instead of being but little removed from its normal position. If you attempted to restore the foot in No. 3 merely by dividing ligaments and tendons at the back of the ankle joint, you would have to produce a decided calcaneus, in order to bring the anterior part of the foot to its normal level, and the result would be ludicrous.

We must therefore direct our efforts to restore the distal part first.

In dealing with cavus or marked contraction of the plantar fascia, as in this, we are told to divide it subcutaneously; this is quite ineffective, except to a very small degree, and is only useful in very mild cases. In such a case as this, Illustration 1, or in that of Illustration 3, much more thorough methods must be adopted.

The plantar fascia is not a string passing from point to point; it is a broad structure of several fasciculi and numerous diverticula which pass down between the muscles to join the tarsal and metatarsal ligaments.

To deal thoroughly then with the shortened fibrous tissue of the sole, we must proceed thus:—

A curved incision convexity upwards is made from the scaphoid tubercle to the metatarso phalangeal joint. This incision is carried outwards across the sole of the foot below the plantar fascia of the muscles beneath it, working your way through with periosteal elevator and knife, a blunt tenotome and finger, until the plantar fascia is quite free from the muscles and intermuscular septa beneath. The edge of knife is then turned towards the surface and the plantar fascia incised thoroughly. I used to advocate the dissecting out of the plantar fascia and then excision of a broad band, but I found that frequent sloughing of the skin resulted, so I abandoned it.

Next, the abductor hall is stripped off the metatarsal bone and an elevator is introduced between it and the bone, and a passage is again worked across the foot—this time between the muscles and bones. Many unnamed fibrous bands will be met with—not the less strong and resisting—which can be cut through with safety, as we are far away from nerve or artery, so long as we do not exceed the limits of our incision. I find a pair of blunt scissors and a strong finger the best for this work, though a blunt tenotome must be used occasionally, as in dividing the long and short plantar ligaments.

Having thus divided all resisting fascial and ligamentous obstructions in the sole, we can unfold the foot and now turn our attention to the tendo Achillis. Very rarely it will happen that the plantar flexors will call for division, as in No. 1; if this be the case, they should be divided at their distal attachment, or above the ankle.

Having thus restored the foot, we must next persuade the dorsal flexors to work, and we must never allow them to be overstretched until they are acting strongly—a point well brought forward by Tubby and Jones. The foot is put up in plaster for three days, and then the patient is given a boot with two steel uprights with toe-raising spring, and a stop joint at the ankle which will prevent the foot from dropping below a right angle. At night the boot can be exchanged for a night shoe.

Clawed toes.—Clawed toes, arising from infantile paralysis, are often attributed to paralysis of the interossei. This erroneous doctrine was formulated by Duchenne, who put it forward because he was unable to obtain an electrical reaction. In a normal foot this reaction is difficult to obtain, and when the big opponents of the interossei are strongly contracting it is almost always impossible to get even a very slight result. In two ways I have proved that the interossei are not ineffective—(1) by dissection, as in Illustration 2, when they were hypertrophied; and (2) by stimulation of the muscle after the clawing has been reduced. This condition arises from a foreshortening of the foot, due to first a lengthening of the distance between the attachments of the dorsal muscles, and consequent contraction of the plantar muscles, and then as the dorsal muscles recover they dorsiflex the foot against their opponents. If you take a dissected specimen and pull hard on both sets of muscles you will produce this condition, according to the strength of your pull. Clawed toes are always associated with some cavus, and, in addition to the method above described for reducing "cavus," we must divide both the dorsal and plantar tendons close to their distal attachment and place the foot in a restored position in plaster.

Talipes calcaneus generally occurs with more marked paralysis accompanying it—*vide* Illustrations 6 and 9. Here we not infrequently have to employ tendon transplantation, or even arthrodesis. Tendon transplantation is as loudly praised in some quarters as it is condemned in others. Its æsthetic effect is generally good, but its utility is not very widespread. We can take a strong tendon, or part of it, and apply it to a weakened tendon; but it is better, if practicable, to lift the muscle with its periosteal attachments and transfer its pull in the direction of the paralysed muscle.

If we have to apply tendon to tendon, I prefer to freshen the opposing surfaces and secure with lateral sutures.

Here we get a much closer union than by the methods of Nicoladoni or Goldthwait, and the pull of the transferred tendon is more in the direction of the re-enforced muscle. Where possible the distal end of the re-enforcing muscle must be united to a neighbour, so as to maintain some of its old activity.

Every case must be considered on its merits; but we must take care that in desiring to bring about an æsthetic effect we are not hampering instead of helping our patient. The peroneus longus, for instance, is one of the most important muscles in the foot, and I cannot conceive of a case that could be benefited by its complete transplantation.

To go back to *Talipes calcaneus*: Here the brunt of the mischief has fallen upon the posterior set of muscles. The anterior may be contracted, as in No. 9, or placed as in Nos. 1 and 6. Where the anterior are active, as in No. 9, but little reposition can be effected, as a glance at the skeleton will tell you.

The chief obstacle is the position into which the os calcis has been drawn or dropped. In advanced cases it is impossible to replace the bone No. 9, but in slighter cases we may effect much by dealing with the structures in the sole, as above described, only our efforts must be placed more posterior. Besides dealing with the plantar fascia and ligaments, we must also cut off the sole muscles inserted into the os calcis. If we cannot then replace the os calcis, cut off its original posterior end—excise as much as we think fit—and apply the original posterior end to the plantar surface of the bone. If it is considered desirable to divide the dorsal muscles, this should be done at or near their distal attachments.

A CASE OF BILATERAL HERPES ZOSTER, INVOLVING AN EXTENSIVE AREA OF THE BODY.

By C. S. WILLIS, M.B., M.R.C.S., D.P.H.,
Principal Assistant Medical Officer of the Government of N.S.W.,

AND

G. R. P. HALL, M.B. C.M. (Syd.).

WE bring this case under your notice in connection with the discussion on Infantile Paralysis, as there is a close resemblance between that disease and Herpes Zoster. In the former disease the anterior cornua are mainly affected, whilst in the latter the posterior nerve roots suffer. Further, the possibility that both diseases are due to the same micro-organism has been suggested.—R. P. Garrow, Maryport, Cumberland. B.M.J., 18/3/11, p. 621.

The history of this case is, briefly, as follows:—

R.L.D., æt. 26, married, one child.

Employment: Book-keeper in a book-shop, necessitating his handling large numbers of periodicals.

Onset of illness: 3/5/11. Began suddenly with pain in stomach, vomiting, and "dry retching." He had some headache, and felt feverish and languid and off his food for the next two or three days.

Seen by one of us (G.R.P.H.) on 5/5/11. Temp. then 103 deg. F., and a few vesicles were present in front of right side of neck. Seen by both of us on 8/5/11, when the temp. was 100 deg. Fah., and the condition, imperfectly shown in the photos. handed round, was found.

A typical Herpes Zoster was present, distributed in the following regions:—

Face: The forehead above eyebrows on both sides, and both cheeks.

Neck and trunk: The whole of the right half of neck and shoulder in front of sterno-mastoid muscle. The rash here extended on to upper part of chest, and also across the middle line to a slight extent. At back of neck, on right side, near middle line, there was also a small area of rash, about 1½ inch by 1 inch in extent.

Arms: Rash was present on both arms extending from about the junction of the middle and lower thirds of upper arms to wrists. In both cases the rash was denser over the fronts of the elbows and wrists. On the right arm the rash was distributed over the outer and anterior aspect of lower third of upper arm, and on the inner side of anterior aspect of forearm. On the left arm the inner and anterior aspects of lower third of upper arm were affected, and anterior aspect of forearm. The hands were not affected.

Legs: On right leg rash was present over lower two-thirds of anterior and inner aspects and nearly whole of posterior aspect of thigh, and slightly on upper part of calf. On left leg rash was on the lower half of anterior aspect and the greater part of posterior aspect of thigh, and slightly on upper part of calf.

The rash was also present to a slight extent on both gluteal regions.

In addition, the conjunctivæ of both eyes were inflamed and excreting pus. No vesicles or ulcers were detected.

This patient was also seen by Drs. A. W. Campbell and D. Thomas, who agreed in the diagnosis of Herpes Zoster.

The nerve roots affected appeared to be both trigeminals; posterior roots of the right third and fourth cervicals; posterior roots of the sixth and eighth cervicals and second dorsals on both sides; and the second and third lumbars and second and third sacrals on both sides.

The patient informed us "the rash first appeared over a small area near front of neck on the evening of the 5/5/11. Next day it rapidly developed, first on the neck and right shoulder and front of chest; then on the thighs; and finally on the arms. The rash was fully developed on the morning of 7/5/11. His general symptoms disappeared on 6/5/11, and after that he felt perfectly well."

During the time the rash was present there was some soreness over fronts of both wrists; otherwise the rash was not preceded, accompanied, or followed by pain.

This man denies ever having had syphilis, and declared "he had not taken arsenic or medicine of any sort for five years." He is also practically a teetotaler.

The patient was seen again by one of us (C.S.W.) on 9/8/11, when the following statement was elicited from him:—"Rash began to fade on 14/5/11, and had quite disappeared on 21/5/11. For some days after the disappearance of the rash there was loss of sensation over the anterior aspect of both thighs, and during this time his gait was so affected that, at his place of business, he was made fun of for the way he walked."

On examination it was found that the rash had left some pigmentation of the skin, notably over the anterior aspect of both forearms.

Dr. D. Thomas informed us that about the time this man was affected he (Dr. Thomas) saw other cases of Herpes Zoster (affecting small areas only) amongst the people living in the same neighbourhood.

The points of interest about this case are:—

(1) The extensive and bilateral distribution of the rash. This is strong evidence in favour of its being due, at least, to a general infection.

(2) Its occurrence about the same time as other cases in the neighbourhood—evidence in favour of the infectivity of Herpes Zoster.

(3) The fact that the three branches of the trigeminal nerve were involved, and that on both sides.

DISCUSSION.

Dr. Stewart McKay said: That he considered that many of the deformities that the surgeon has to deal with in connection with cases of Infantile Paralysis, could have been prevented. They were due to the neglect of the medical man attending the case, and the early use of splints would help to prevent these deformities in the future.

With regard to operations on the bones, joints, and muscles, he had now performed some two hundred operations in these cases, and he considered that the results in many cases were quite good when the paralysis was below the knee-joint; but he was not at all satisfied with the results when the muscles above the knee-joint were affected to any extent. For that reason he had begun to select cases, and to practice nerve-grafting on them. The nerves that he had selected were the anterior crural, and the external popliteal. He took the deeper portion of the anterior crural, and brought it out behind and grafted it into the sciatic. In other cases he grafted the external popliteal into the internal popliteal. It was too early yet to say what the results would be.

Dr. Flashman expressed regret that he had not been present to hear Dr. Mackenzie's paper, but he gathered from the discussion that Dr. Mackenzie maintained that he was able to restore the function of paralysed limbs by focussing his attention on the muscles. No doubt considerable tension had been felt by those listening to some of the previous speakers. It seemed to Dr. Flashman that it was obvious that a misunderstanding had arisen. During the first shock of the disease a great amount of the motor region of the cord is damaged by the inflammatory condition and by the toxins circulating around the neurone. The immediate result was extensive flaccid paralysis. In a few days, as this acute condition passed off, many of the threatened neurones survive, resume their normal functions, and the muscles to which they are attached recover their action. Some of the ventral horn cells are destroyed. Axones from neighbouring healthy neurones, whose own axones may have been interrupted or destroyed, grow out into the empty myelin sheaths. After a considerable period, when these axones have reached the periphery, recovery of function of the paralysed muscle is made possible, so far as the nervous connection is concerned. If the muscle has been maintained in a healthy condition during the time that the axone has been growing out, the recovery of function will take place gradually and certainly. If, on the other hand, during the time the axone has been growing out, the muscle becomes overstretched and loses tone through the action of the opposing muscles. no recovery of function may take place, even when the neuronic connection becomes complete. Is it not possible that Dr. Mackenzie has come across a number of these latter cases? He has found cases with overstretched and toneless muscles, but with muscles whose nervous connection have been repaired. Certainly, if such were the case, all Dr. Mackenzie would have to do—but it must be done carefully and thoroughly—would be to centre his whole attention on the muscle with a view to restoring its tone. I expect that Dr. Mackenzie has had considerable success in carrying out this natural treatment, but owing to the storm of indignation that he has aroused in some quarters, he has either used wrong expressions, or his expressions have been misunderstood, and his critics have conceived that Dr. Mackenzie believes that poliomyelitis anterior acuta is a muscular disease, independent of trouble in the central nervous system. At the present day this idea of the disease being muscular *ab initio* is so absolutely inconceivable that one must immediately acquit Dr. Mackenzie of holding it, and must believe that the criticisms so strongly directed against him have been based on a misunderstanding of his real views.

Dr. MAX HERZ: The subject of infantile paralysis has everywhere of late claimed the attention of all concerned in public health. Large epidemics of this terror to our children have visited many countries, and left behind them the hope of the future slain or maimed. And yet there was something good in a thing evil. Material has been furnished to observers and experimenters, and as a result of their researches our thoughts and ideas on infantile paralysis are to-day in the melting-pot; and we stand on the verge of having fulfilled an old ambition and devout wish to see and get under our control the terror-bringing virus.

The human brain often works in analogies. And it is passing strange to observe how fertile and full of fruit the thought was that infantile paralysis showed a marked resemblance to rabies in its choice of the cord as the point of assault, even in its morbid anatomy, as the marrow lesion presents the perivascular infiltration characteristic of rabies. The recognition of this resemblance, superficial as it may seem to be, furnished the torch to light up things unknown; it led Flexner and Lewis and Romer to undertake inoculation experiments on lines which had been followed up successfully by the Pasteur School in the case of lyssa. Emulsions were prepared from brain and spine marrow of children who had succumbed to an attack, and introduced into the cerebrum of monkeys. And the result surpassed the boldest expectations. After an interval of four (the shortest) to thirty-three days (the longest), the infected ape became sick. A quick and profound change occurred in most cases. They lost their merriness, and grew limp and seedy. After one or more additional hours paralysis set in. Other animals showed a more gradual onset. The extent, degree, location of paralysis varied. In more than half of the cases the lower limbs were thrown out of action, while other showed paralysis of the upper extremities and medulla; some recovered, but most retained residues. In short, the history of human poliomyelitis is repeated in all its details and varieties in the infected animal. The marrow of those which died of an artificial poliomyelitis proved fatal to another set, and the continuous transmission is not only feasible and possible, but has actually been carried out through twenty and more generations. It was proved, further, that a monkey which had battled through a more lenient attack of poliomyelitis was immune against an infection which killed a sound control animal. Römer could produce, by injecting a small dose of virus containing emulsion, immunity in monkeys.

The virus has not been seen by human eyes, but our laboratories study it, and, bit by bit, tear off the mysterious veil surrounding it. It is filtrable, like the germ of yellow fever, withstands great cold, can be kept frozen for many days—for almost two months—without losing any of its deadly sting. It stands drying for many days over caustic potash, which turns the spinal cord dry and brittle, without suffering a loss of activity. It belongs to the class of highly resistant viri, which includes the micro-organisms of rabies and of vaccinia. We know nothing of the virus apart from its host, through which dissemination can take place. There is no evidence, says Flexner, that any of the filtrable viri live a saprophytic life, the virus having thus far been found only in connection with animals which have been diseased or which acted as passive carriers of the infection. The virus, when once discovered, will most likely be demonstrated not only in connection with persons who suffer from poliomyelitis, but in those who are passive carriers of the disease. Whether this infantile plague constitutes a contagious complaint is yet a question.

The virus finds its entrance into the human body, in all probability, through the naso-pharynx, and perhaps its exit also. The diplococcus intracellularis, the originator of the cerebro-spinal meningitis, enters through the same door, travels through the cribiform plate, and reaches the meninges. It is very likely that the viri can leave the body by the same way, as Flexner succeeded in demonstrating that a diplococcus, injected into the spinal canal, made its appearance in the naso-pharynx. Probably, then, the virus of infantile paralysis wanders by the same route. It is most significant that modern researches prove that an inflammatory process in the pia mater is never absent. It consists in a diffuse mononuclear round cell, probably lymphatic infiltration. This meningitis is most pronounced in the sacral and lumbar regions of the cord, the cervical being next. Its greatest intensity is reached, according to Strauss, on the anterior surfaces of the cord, whence it follows the pial process into the depths of the anterior fissure and along the central vessels. The mononuclear cell infiltration continues also around the other vessels of the cord. The posterior root fibres, and the arachnoid membrane covering the spinal ganglia are likewise infiltrated. The virus then enters the cord by way of the perivascular lymphatic vessels. The disturbances set up by it here, in the perivascular and adventitial tissue are considered by some as of the greatest importance, almost as the chief event of this disease. The infiltrations are collections of small mononuclear round cells, which lie in the adventitial lymph spaces, the capillaries showing the same infiltration. The anterior horns are more affected than the posterior, probably because of their richer vascularity. The vessels of the cord, medulla, pons, basal ganglia, even of the cerebral cortex, are dilated and engorged, the capillaries being extended to more than twice their normal calibre. Hæmorrhages in the grey matter, although not extensive, demonstrate the involvement of the vessel walls. The poison of the virus enters the tissue of the cord itself, and sets up inflammation, causing irritation and destruction of the living cells, and the reaction of the organism against the intruders consist of hyperæmia, œdema, concentration of lymphocytes. Many pathologists look upon the vascular and interstitial changes as the primary alteration, regarding the affection of the ganglia as secondary, ganglia being only attacked when in contact with the inflammatory process around the vessels. But they have to admit that the virus shows an affinity to the ganglion cell, and a special affinity to the anterior horn ganglion cell, which, by way of blood and lymph vessels, the virus reaches first, and with a yet unabated vigour. The fact remains, however, that in experimental poliomyelitis the virus introduced into the body intracerebrally attacks, first and mainly, the lumbar cord, and we have, for the want of other reasons, to assume a special affinity or a lessened resistance on the part of the ganglion cell. All levels of the cord are hyperæmic, and the inflammatory œdema spreads out wide through the whole cord, interrupting the conductivity of nerve fibres; its subsidence restores the affected nerve tissue to their function. Remarkable among the lesions of the internal organs is the acute enlargement of the solitary follicles, Peyer's patches, the acute inflammation of the mesenteric glands. Does the gastro-intestinal tract provide another mode of entrance for the virus; or is this inflammataion a sign of the elimination of the toxin? The symptoms of anorexia, vomiting, diarrhœa, often observed in connection with the fact that the changes are found very early—three hours after onset of the attack—suggest that the virus finds a second door of entrance through the intestinal walls.

The clinical appearance of our paralysis must needs vary according to the pathology. There is the classic picture: the rapid onset with malaise, fever, vomiting, quickly followed by an irregularly distributed paralysis of the motor-spinal nerves, the cranial nerves seldom participating; after a short interval a general improvement begins, with restoration of functions, but not without leaving residues behind. Another form has, as its most significant feature, the meningeal symptoms, the retracted head, the rigid back, delirium, intense pain and hyperæsthesia of various regions of the body, joints, &c.; but a menigococcus cannot be found. The symptoms disappear in a few days, as against the long duration of meningitis. Paralysis or paresis of one or the other muscle will rarely be missed. Yet another type sails under the flag of periphereal neuritis, with extremely tender limbs and soreness in muscles; here also the flaccid paralysis is seldom absent. The resumption of function is chiefly due to the subsidence and absorption of the inflammatory œdema which interrupted the conductivity of ganglion cells and nerves. Some epidemics, as that observed by Krause, showed gastro-intestinal symptoms in about 90 per cent, accompanied by fever and heavy sweating; in others, as in that seen by Müller, of Marburg, the intestinal signs were exceptional, initial angina. bronchitis, and broncho-pneumonia taking their place.

The modern researches have taught one thing: that the names so far given to the disease do not fully express its character. Our paralysis is by no means only infantile; adults are attacked, although fewer in number. I have observed a gentleman of 30 years being infected, and have at present under my care a gentleman from Gunnedah (N.S.W.), who, in his 59th year, fell a victim to the disease in January of this year. The term of anterior poliomyelitis is too narrow, as it is really a diffuse meningo-myelitis, even an encephalitis. To call it, as has been proposed, enkephalo-meningo-myelitis, would be a very vague definition, besides the ugliness and jaw-breaking qualities of the word. Krause seems to have proposed the happiest solution—acute epidemic paralysis—and I recommend the name to your consideration.

Thus far the modern researches have widened our knowledge. I hesitate considerably to say that about a pamphlet issued in Melbourne by Dr. Wm. Mackenzie. Although, after what has been said, silence could hardly be interpreted as consent, I will give the booklet a few passing critical remarks. The pamphlet suffers most severely from ignoring all literature on the subject. The overwhelming evidence furnished by the startling discoveries of Flechsner, Lewis, Landsteiner, Levaditi, Roemer, Wiesner, is brushed aside with the simple confession of his "non-belief in the ordinary accepted theory of destruction in the anterior cornua of the spinal cord." Mr. Mackenzie seems to consider the changes in the spine as "sclerotic changes, which probably do not occur under proper treatment." And this in the face of the evidence furnished by the morbid anatomy of fresh cases, and of experimental poliomyelitis. What, then, is the pathology of infantile paralysis, according to Mr. Mackenzie? It is very hard to gather his views from his book. But I believe I am right in stating that he thinks it is a muscle complaint, pure and simple. To quote him:—"As regards a definite specific organism as the cause of this disease, we have always to remember a toxic agent as causative—*e.g.*, beri beri and plumbism.

The disease has frequently followed one of the infectious fevers." He thinks, then, of dietetic causes, of food-infection. What then is, I ask again, in Mr. Mackenzie's opinion, the pathology of infantile paralysis? Apparently some toxic agent which attacks the muscle. A toxic myositis which creates paralysis? A new thing! For plumbism is a neuritis. Mr. Mackenzie felt somehow that he owed the world some evidence, and went about to furnish it. He inoculated a monkey with a saline emulsion of small portions of the tibialis anticus and extensor digitorum longus of an infected limb of a boy 3 years old, with a two days' history. The inoculation was made into the leg of the left lower limb of a young monkey; the exact spot is not communicated. Suppuration set in, caused by staphylococcus aureus. The limb was noticed to be helpless four days after the inoculation, the animal dragging its limb in the endeavour to escape. To quote exactly:—" Later, contraction of the hip and knee-joints, with flaccid ankle and dropping of toes, were noticed, as seen in the illustration. The paralysis was marked four weeks after its recognition, when an autopsy was made; and for three weeks no evidence of pus had been noticed, and the contractures of the knee required considerable force to overcome. All the muscles of the limb were markedly attenuated, especially the two vasti and the rectus femoris." "Nothing was found in the spinal column." This is all that Mr. Mackenzie offers us as sufficient and convincing evidence of a new theory. His notes are, to say the least, certainly not exact and thorough. A paralysis is spoken of, but it is not detailed, the names of the paralysed muscles not being given. The monkey drags his leg. Well, there was a suppuration, and there was in all likelihood pain, and so the poor animal dragged its leg. We are referred to the illustration. And this photo. is, to my mind, which is quite impartial, absolutely unconvincing. To put it that mild way, it offers itself easily to an interpretation quite differently from that given by Mr. Mackenzie. This monkey picture does not show that the left ankle drops any more than the other, and the knee and hip flexors are certainly not paralysed. It shows essentially the position of an animal with a painful leg, and no more. That the autopsy found an attenuated quadriceps is not a matter for astonishment, as this muscle always suffers atrophy in any slight leg affection. Mr. Mackenzie's evidence has been weighed in the balance and found wanting. His proofs are insufficient and useless. His confession of his non-belief remains a subjective utterance, not substantiated by any scientific observation or facts. They are words, words, words! He promised to give us bread, and gave us a stone. His little pamphlet will not stop or turn the rolling wheel of modern research; the wheel will go over it.

The discoveries of the last years have, so far, not given us any help in the treatment. But here, too, things are not of so hopeless a fashion as they were a decade ago.

In epidemics one is well advised to keep a watchful eye on the naso-pharynx, as this part of the body, with all likelihood, plays an important role in disseminating the virus. As the infectiousness of the disease, in spite of Mr. Mackenzie, is proved beyond doubt, and the contagiousness is still a matter of discussion, it is wise to take resort to isolation and other methods usually employed in infectious diseases. The key for the treatment of the acute period is: rest; as little movement as possible, so as not to irritate the meninges and cord. The plaster of paris bed, moulded to the little body, provides the best means for this purpose. In the period of recovery, *i.e.*, for the six, nine, twelve months after the onset, the chief and main endeavour of everyone must be to avoid deformities, to avoid overstretching of muscles. These principles have long been proclaimed, but they are seldom adhered to. A splint is the best method, but not one of the kind usually provided by the instrument-maker, but one made over the plaster cast of the paralysed limb to secure a perfect, glove-like fit, either of leather alone, or reinforced with steel, not too heavy, just strong enough to fulfil the purpose. These supports keep the foot in normal position, will take the weight of the limb off the muscles, and prevent them from becoming overstretched. This was known and practised, by the way, long before Mr. Mackenzie published his pamphlet, so that the good contained in the book is not new (and the new is no good). His zero-position is of questionable value. It is essentially the position of the greatest relaxation of the muscle, and if kept there by a splint, means simply that no weight of a limb is able to drag it down and stretch it. If Mr. Mackenzie feels it incumbent to invent a new name for a thing old, I will not rob him of this discoverer's pleasure.

But that a weak muscle will only work in this relaxed position is not always correct. Certainly an atrophic, paretic muscle is not able to lift a weighty limb, but to contract at all it must have its tonus, and this tonus in an overstretched muscle is only restored when the muscle is in a slight tension, in other words, when the joints are in a flexed position.

I turn now to the third period, when, after nine, twelve months from the onset, the paralysis has become complete, and nothing but splints or operations will give back the use of the limb.

I am again here in opposition to Mr. Mackenzie, who confesses the belief that "arthrodesis, tenodesis, joint erosion and excision, tendon transplantation will be largely, if not entirely obviated." Scientific reasons for this belief are not given, and this faith remains, like all, a matter of sentiment. But I must confess that Mr. Mackenzie is easily pleased with his results—too easily. He applies a caliper splint to a flail limb, converting it into a rigid piece, and pronounces, with pride in his heart, that on the same day the patient walks without support. Although he wears his splint all the while, yet it is called "without support." It is hard to take such argumentation seriously. The doctor is apparently pretty firmly convinced that, with exercises from the zero-position, every quadriceps can recover. But I have grave and severe reasons to believe that Mr. Mackenzie labours under a great illusion. He wants us to "turn the patient completely on the opposite side, and so alter the leverage, and our patient will be able to swing the limb from the hip in one piece, *i.e.*, with full knee extension." Certainly, he will do that. But has it never occurred to Mr. Mackenzie that this is no quadriceps action at all? It is a flexion and extension in the hip joint. The patella looks sideways, not upwards; the gravitation strikes the knee in the axis from lateral to medial. The configuration of the knee, the lateral ligaments and capsule prevent it from being bent. Turn that patient back, a little only, so that the underleg can drop, and you will see how quickly it does fall. The case photographed on page 17 is called an ideal recovery of left quadriceps paralysis. And yet one cannot let it pass as that. The boy lies on his right side; the right gluteal region is visible. That, firstly, proves that the boy is not lying on his back, straight and flat; secondly, that his movement is not a flexion in the hip with an actively extended quadriceps, but that it is a combination of abduction and flexion of the hip, the knee being in a position which puts gravitation out of action, when the knee possibly cannot flex. I fear it is a vain desire to wish to avoid surgical operations in the treatment of infantile paralysis. Although, according to Dr. Mackenzie's theory, all muscles really should recover from the poisoning by a toxic agent, he himself has to take to the knife. And, again, I fail to agree with him. He splits the fascia which covers the muscles, with the idea to give the attenuated muscle room to expand, to relieve it from pressure. The thought that one soft tissue could prevent another from growing is, at least, novel; but really it will not do! We know that even the firm, hard bone becomes corroded, atrophied, when an aneurysm presses against it, and a fascia is suspected to stop the growth of a muscle, whilst the experience of every day proves the contrary. Although neither theoretical nor practical arguments are brought forward in support of this strange idea and proposal, I had repeatedly an opportunity to split the fascia when doing the arthrodesis operation, about which more will be said presently. No result whatever was seen after from this proceeding; the inactive muscle remained inactive, the paralysed paralysed, and the dead did not awaken, although the split in the fascia was meant to throw open again the door of life.

There remain, then, the approved methods of transplantation and arthrodesis, often combined, often to be employed in the same limb.

But, before any operation is attempted, it is indispensable to remove any existing deformity, and, by means of wrenching, we can nearly always achieve this end. An endeavour to correct and transplant at the same time courts failure!

The success of a transplantation depends on the muscles at our disposal, and on the technique of the operator. The choice among the surviving muscles to be transplanted must be a judicious one. The questions are: Which of the paralysed muscles have to be replaced to restore the use; and which of the survivors can be spared without impairing and harming the function of the limb? And is it possible with the muscles at our disposal to restore the function to the satisfaction of the patient? This last question receives sometimes too ready an answer. There are, for instance, a few muscles left in a foot, the calf and the flexor hallucis longus. The enthusiasm might drive the eager surgeon to use this flexor instead of the tibialus anticus, and sew, perhaps, half the Achilles tendon into the peronei. It sounds all very well; but his will be no result; because he asks too much of the two muscles. Our patients want feet which hold them securely to the ground, which do not turn in or out, but stand straight and carry the weight well. And in many a case an arthrodesis is more indicated than a transplantation, or both should at least be combined. For transplantation, we use the muscle with the less important function to replace the more important ones. It is of greater value for the patient to move his feet than his toes; of greater utility to flex his elbow than to straighten it with the aid of a muscle, as gravitation suffices for the movement. The choice among these muscles must be judicious and careful, with an eye on the future function.

The other important factor is the technique. How should the transplanted tendon be fixed? There is the method of grafting tendon into tendon with its two divisions into active or descending and passive, or ascending transference. Or we choose new points of attachments, and sew the tendon to the periosteum, or fix it into the bone. My experience made me a firm confessor of the last method. But, I admit, all methods have their uses, meeting certain requirements. Often all are employed in one and the same case and operation. We may also divide a tendon and graft the part split off somewhere else. But to secure success, it is, in my experience, essential to provide a like function for both parts. It seems to me to be ultra vires of a muscle to supply power for two antagonistic movements, to act, for instance, as extensor and flexor.

The fixation of the tendon claims the greatest care of the operator. There is a law in muscle physiology which it is well to remember. It says: The effect and direction of a muscle contraction are ruled and guided by that piece of tendon which extends free from one part of the body to the other. For transplantation this means: The part determinating the direction of the new movement is the lower part, the tendon. It is immaterial how the muscle-flesh runs. It is thus most important to let the new tendon run in the direction in which the intended movement is expected to go. Another problem of the tendon fixation is: sew securely, without damaging the sinew, and prevent adhesions. The tendon—and this is of the greatest importance—the tendon must be fixed in a way procuring, as near as possible, the normal tonus of the muscle. And this tonus is to be secured by the tendon suture. Such suture (we always use silk) should never go around the whole tendon. The circulation of a sinew is not very good at any time, and a ligature passed around it brings quickly gangrene, preventing healing by first intention. To prevent adhesions of the sinew to the skin or underlying tissues, a proper sheath should be prepared. And this leads to another point which must not be lost sight of: the tissue through which the transplanted muscle-tendon is to be conducted. It should be fat wherever that is possible. For fat or connective tissue or muscle are themselves movable, and adhesions here are not of such hindrance; but when a tendon becomes adherent to a firm fascia or bone, this connection will have a pernicious influence upon the movement, as it will make it impossible, the tendon having no free play; the transplanted sinew in such a case will be, and can only act as, a ligament. To my mind the time of bandage fixation usually employed (six to eight weeks) is far too long. It invites the tendon to become firmly knitted to its surroundings. If deformities are corrected first, and transplantation follows, there seems and there is no need to preserve the position for such a time, and court adhesions.

A fixation period of three weeks is ample, in my experience.

The transplantation of nerves is a young shoot on the tree of paralysis treatment, and it has come to stay. Only infantile paralysis itself will, perhaps, see less of it than other kinds—paralysis after traumatism, for instance; for it must be remembered that poliomyelitis very seldom, hardly ever, attacks one nerve wholly and solely, but only parts of one nerve, or parts of different nerves. It must not be forgotten that the central part in the anterior horns is attacked, not the peripheral nerve. To give nerve transplantation that position in our plan of healing which it is perhaps entitled to, one chief question has to be answered first, and that is: What is the topographical anatomy of a nerve? Where, in a section, in a cross-cut through a nerve, lie the motor fibres for the different muscles; where the sensory ones, so that we may know which part to replace, which one to take for grafting without doing harm? Until this anatomical question is answered completely, nerve transplantation in cases where not the whole nerve is affected, is merely guess-work and a matter of chance and luck.

But what is to be done in cases with very few or no muscles left? The answer is: Albert's Arthrodesis, the stiffening of joints. I have tried various ways, have fixed silk ligaments in the foot, across the knee, but have always come back to the excision and actual stiffening of joints. And I now put before you a device which has never disappointed me during the last twelve months. No nails or staples are employed. We use bone pegs, with their periosteum, which are sawn off the patient's own tibia. These pegs are used to fix the knee; they are employed to fasten the totally paralysed flail foot. We drive it through the calcaneus into the astragalus, fix it as a bridge over the Calcaneo-cuboid, or Astragalo-navicular articulation. We do not stiffen the joint between tibia and astragalus; to overcome the equinus position, we sling the extensor digitorum longus and tibialis anticus on silk threads, and sew them to the tibia periosteum; we prefer to leave the movement in this joint, as it gives a little spring to the foot, but we lay greatest stress on the absolute fixation of the lower joints, so that the patient cannot turn over on his foot. We have not met a failure with this technique so far; the healing goes on without the slightest disturbance; the position of the foot is secure. X-ray photos prove that actual growth is proceeding from the implanted periosteum, whilst the bone part becomes gradually absorbed, being replaced by the newly-formed. I go so far to say that, with this technique, the paralytic deformities of the feet have lost their terror for us. And as we are always very careful not to touch the epiphyseal lines, we cannot see any age limit.

The advances in the surgery of the so-called infantile paralysis have been rapid, the success and results gratifying. They have given us the means to place unfortunate cripples once more upon their feet, free them from crutch and splint, and make useful citizens out of objects of pity and charity.

Dr. C. S. WILLIS: In November, 1909, I was sent by the Department of Public Health to investigate an epidemic of infantile paralysis on the Northern Rivers of New South Wales. The following interesting points were observed:—

1. From end of August to second week in November, 1909, the number of cases was as follows:—Ballina, 16 cases, no deaths; Broadwater (20 miles from Ballina), 6 cases, 1 death; Murwillumbah (about 70 miles from Ballina), 2 cases, no deaths.
2. The first case (a Ballina case) occurred in the same house and in the same family in which a child had died three years previous of infantile paralysis. This I looked upon as more of coincidence than an ætiological factor.
3. Practically all the cases in Ballina had business communication with one block in that town. This communication was in the nature of obtaining fruit, sweets, &c., from shops in this area. Three cases occurred in two sweet and fruit shops in this block, and the father of two other children attacked with infantile paralysis also kept a shop in the same block. Further, most, if not all, of the cases at Broadwater obtained food supplies directly or indirectly from the same area in Ballina.
4. The Murwillumbah cases appeared to have no connection with either the Ballina or Broadwater cases.
5. Sex and age distribution:—
   (a) About an equal number of males and females were attacked.
   (b) Nearly half of those attacked belonged to the age period of two-three years.
6. One case presented partial right-sided facial paralysis. One-sided facial paralysis has, of course, been observed in other epidemics, notably that at Westphalia, in the spring of 1909.
7. The question of interest, from an epidemiological point of view, is, how did the infecting agent reach Ballina? Careful inquiry elicited the information that none of the earlier cases had been out of the town for some months before the onset of the illness. Three possibilities presented themselves:—
   (a) The infecting agent remained dormant for three years in the family or surroundings of the first case. This is improbable; also in other parts of New South Wales cases of infantile paralysis occurred at this time.

(b) Some "carrier" took the infection to Ballina. I was unable to find any reason to suspect anyone, but the non-finding of a carrier, considering the difficulties, proves nothing.

(c) The infection was carried on some article of diet.

8. As regards pathology of infantile paralysis, Past-Assistant-Surgeon Wade H. Frost, Public Health and Marine Hospital Service, U.S.A., states: "For the past twenty years the essential primary lesion was considered to be (inflammation and) degeneration of the motor cells of the anterior cornua of the cord. This misconception arose from the fact that the earlier pathologic studies were made chiefly on cases that had long since passed the acute stage. Yet, in the past twenty years, there has been ample evidence, both clinical and anatomical, that in the acute stage characteristic lesion is an interstitial inflammation, not confined to the motor area of the cord. Acute anterior poliomyelitis must be recognised as a general infection producing characteristic lesions in the central nervous system, viz., congestion, infiltration, and œdema of the cord, brain, and leptomeninges."

9. Just a few words regarding preventive measures. The probability that infantile paralysis is a contagious disease has been sufficiently demonstrated to render preventive measures imperative. It would appear that this disease should be made notifiable, and proper isolation and disinfection insisted on, as in other infectious disease. This would bring home to the lay mind, and also medical men, the fact that this disease is contagious. The value of isolation was shown in the Northern Rivers epidemic. At Murwillumbah both of the cases were isolated, and the spread of the disease was prevented. (Onset of cases 15th and 26th October; visited by me 11/11/09.) At the two other towns practically no attempt at isolation was made, and a comparatively large number of cases occurred.

I think, Mr. Chairman, it would be advisable if the combined Section expressed an opinion as to whether the notification of this disease should be compulsory. How long should patients be isolated? This is a difficult question to answer, but I think rigid isolation should be enforced, at least until all acute symptoms have passed off. It would also appear advisable to exercise some supervision over the movements of contacts, and especially should children contacts be prevented from attending school for a period, say, three weeks. Also some antiseptic treatment of the mouth and throats of contacts might act beneficially in helping to prevent the spread of the disease.

Dr. R. B. WADE: With such a large subject in front of one it is difficult to limit oneself in this discussion; but there are two or three points on which I should like to touch.

The actual causative agent in the paralysis is one of interest, and should be considered from the important point of view of our treatment of the immediate paralysis. The generally accepted view is that there is an invasion of the cord by the organisms, with resulting inflammatory changes, consisting mainly of a dilatation of the vessels, accompanied by a perivascular infiltration of cells around them, small hæmorrhages and serum exudation, and that the pressure of these either destroys the anterior horn cells absolutely, or else disables them and throws them temporarily out of action, so that at the time of the acute attack they lose their power of functionating and transmitting motor impulses, and that as the exudate and products of inflammation are absorbed the compressed cells regain their functional power again. That inflammatory exudate has this power of holding in abeyance the function of the motor cells is pure theory, and has, as far as I know, nothing to back it up. Again, the period during which rapid recovery from the primary paralysis may occur, is any time from two to eight weeks after the onset of the illness, as I have never seen the acute febrile condition last longer than ten days, and generally not more than five; while the process of initial muscular recovery may go on for at least six weeks after all signs of the infectious condition have passed. Our experience of inflammatory conditions in other parts of the body go to show that when the cause is removed—and this is shown in the case of infant paralysis by the cessation of the fever—that the inflammatory matter tends to disappear rapidly in a few days, or week at most; and yet here we have, according to the pressure theory, it persisting for, perhaps, six weeks.

Similar changes may be seen in the cord in tetanus and rabies, and yet the affection of the motor cells, as evidenced by the preliminary irritation, must be of a nature different to that of pressure, and is generally considered to be toxic.

The affected muscles in the acute stage are always tender to pressure, suggesting an inflammatory condition of them pressing on the sensory nerve endings.

Swelling, too, of the nerves has been described, though I must confess that I have not observed it.

The whole picture of infantile paralysis in the acute stage, as it appears to me, is that the paralysis is purely of a toxic nature, and that the probability is that, instead of the toxin merely affecting the anterior horn cells, that it also affects the whole motor segment, motor cell, prolongation, and muscle.

Dr. W. F. Litchfield : My experience of infantile paralysis has been fairly extensive, having seen cases during two epidemics in Sydney, besides many sporadic ones. In the spinal cases in my experience, the most striking things are the large amount of improvement that takes place in the initial paralysis, and the great aid that can be given to the cases with residual paralysis by effective splinting. I am an advocate of early splinting, and think the cases should be followed up after the acute stage is over, and the weakened limbs properly supported and surgical procedures adopted when indicated. It is curious to note that, in the 1910 edition of such a widely-read text-book on diseases of children as Goodhardt and Still's, nothing is said on the value of splinting in these cases, the old remedies of electricity, massage, baths, and warm clothing being advocated. Surely in the treatment of infantile paralysis we have passed the gospel of drift and the attitude of pessimism! However, I wish this morning to confine my remarks mainly to the disease as it attacks the higher centres. Children are subject to certain acute paralyses of cerebral origin, of which hemiplegia, acute ataxia, and acute tremor are examples. Until recently the origin of these attacks was not known, being variously attributed to hæmorrhage, syphilis, tubercle, and encephalitis of a septic nature. Strumpel was the first to suggest that many of these cases resulted from a polio-encephalitis of a similar nature to poliomyelitis. Other writers have accepted this view, and polio-encephalitis superior is every day having a wider role attributed to it. The facts relied on to establish the identity of these acute cerebral disorders with anterior poliomyelitis are, the similarity in the ages of the children attacked, the similarity in the seasonal incidence, in the suddenness of onset, and in the amount of recovery that occurs; additional evidence is afforded by the experimental work of Flexner and his pupils on monkeys. The commonest form of paralysis of cerebal origin in children is hemiplegia. The following is a case:—A baby girl, aged one year and three months, was noticed to be feverish one day about noon; at 6 p.m. general convulsions set in, lasting three hours; some hours later the convulsions recurred, but were limited to the left side; next day the left arm and leg were found to be paralysed and flaccid; three weeks later some movement had returned to the limbs, and the flaccidity had given place to spasticity; at the present time—three years after the attack—there is a slight spastic hemiplegia on the left side, and slight mental deficiency. The lesion in cases of this kind is in the cortex on the opposite side to the paralysis. I have here under the microscope, however, the section of the cortex of a child who died from encephalitis in the acute stage. It shows extravasation of red blood-cells and infiltration with leucocytes. The child was eleven months old, had general convulsions, followed by semi-unconsciousness, rigidity of the legs, alternating squint, and marked irritability when moved. *Post-mortem* there was deep hæmorrhagic staining of the cerebral hemispheres, being more widely spread on the left than the right side.

Cases of acute ataxia are less common. The following is a case: —A girl aged 17 months, first seen by me in May, 1910, gave a history of convulsions lasting several hours six months previously, followed by a loss of the use of the limbs, neck, and trunk, but with no apparent disturbance of the intellect. On examination there is no paralysis, but a universal want of co-ordination of the muscles. The child falls over in any direction when sat up or stood up. The child can grasp, but the hands move in an uncertain manner. There is some hypotonia, no tremor, and the eyes are normal. The mother declares that a good deal of improvement has taken place during the last few weeks. Cases of this kind are probably due to a lesion in the dentate nuclei of the cerebellum. Cases of acute tremor in children have been reported by Miller and Leonard Parsons, and, on the authority of Gordon Holmes, the lesion in such cases is in the cerebello-rubro-spinal system. I have not seen any cases of acute tremor, but in the following case the lesion was probably in the cerebello-rubro-spinal system:—A boy, aged 11 years, seen by me on the 7th July, 1911, gave a history of a convulsion two months previously. He remained in an unconscious condition for nine days, and was unable to walk or speak for some weeks afterwards. Latterly, he has improved considerably. On examination there is slight but definite general ataxia of the cerebellar type; the deep reflexes are increased; in other words, there is a muscular hypertonia; there is also a peculiar affection of speech. The latter is slow, deliberate, exact, with a remarkable spacing of the syllables. The intellect is little, if at all, impaired. Cases resembling disseminated sclerosis have also been reported, and it is obvious that scattered foci of inflammation affecting the grey matter of the brain may at times give rise to a multiplicity of symptoms.

Dr. W. J. Hope (Perth, W.A.): Polio-encephalo myelitis acuta—affects the grey matter of the cord, and may extend to the pons and medulla.

It is more common in children, and especially in infants, than in adults; due, probably, to a special vulnerability of the grey matter during infancy to organisms or toxins. No epidemics have been known, but sporadic cases have occurred in West Australia.

Inquiries as to the existence and prevalence of infantile paralysis in Western Australia have produced only the following information:—

Four cases were treated during the past two years in the Children's Hospital, of which paralysis of one or more limbs, resulting from anterior poliomyelitis, was present. In three of these four cases the paralysis was long-standing, and had nothing to do with the condition for which the children were under treatment.

There is no evidence to show whether the original paralysis had occurred in the State or not.

In the four cases there were some paresis of one of the limbs, but it is very doubtful whether this was poliomyelitis or not. Inquiries from the Registrar-General's Office reveal the fact that one death was registered as being due to this disease in 1909, but no other case is recorded during the past fifteen years. Epidemics have appeared in other parts of Australia.

Heat favours, cold weather brings an epidemic to an end.

It would be expected that a microbic origin is in evidence to account for a febrile disease. Micrococci have been discovered which, injected intravenously with rabbits, mice, &c., produced paralysis.

It is held not to have been proved that these organisms are the cause of symptoms produced, but to be in the nature of contamination or accidental invaders. More valuable results have been obtained by injecting into monkeys emulsions of the spinal cord of patients dying from the disease. These injections produced inflammatory changes in the spinal cord, and further transmission from monkey to monkey by intra-cerebral, intra-peritoneal, intra-neural, and hypodermic injections of emulsions of affected monkey's cord.

The non-transmissibility by inoculation of cerebro-spinal fluid from patients at various stages of the disease, shows that the causal agent is no ordinary microbe.

The emulsion of spinal cords causing the disease in other monkeys shows the virus to be of extreme activity, but its nature has not yet been identified, and it is not arrested by the finest filtration.

No cultures have been able to be grown in any of the ordinary media. Drying for seven days, mixture with glycerine, nor with 5 per cent. carbolic acid, does not diminish its virulence.

Flexner and Lewis were able to detect the virus in the nasal and pharyngeal mucus of affected monkeys. They considered that one path of elimination of the virus was by way of the nasal mucosa, through its lymphatic connection with the meninges.

It is probable that infection occurs by the same route in a reverse direction. This hypothesis was tested by scarifying the naso-pharyngeal mucosa and applying an emulsion of a polio-myelitic spinal cord, and repeating it four days later. On the seventh day paralysis of the neck and upper limbs appeared, and the animal died the same night.

The virus of this disease produces rapid and characteristic alteration in the cerebro-spinal fluid and in the central nervous system. With these changes, even after seventy-two hours, no paralytic symptoms had developed. Yet the cerebro-spinal fluid in advance of the onset of paralytic symptoms contained the virus, and when injected intra-cerebrally, set up paralysis in another monkey seven days after injection.

If these observations on the cerebro-spinal fluid be confirmed in the human subject, a valuable means of diagnosis will be secured, and inoculation of a monkey may confirm the diagnosis.

Incubation period—average eight days—from four to thirty-three days.

Mortality in monkeys, 50 per cent.

Mortality in humans, 5 to 20 per cent.

Pathological changes in the nervous system are similar in man and monkey. It is not confined to the anterior cornua; the virus passes on to the meninges, pia arachnoid, vascular dilatation, extending along the pial septa, and in time at anterior horns; also posterior root ganglia and posterior roots; thus accounting for the acute pains found to usher in the disease.

Mode of Infection.—Communicable from one to the other, and through carriers. Transmissible from house to house. Virus hangs to house where disease has occurred.

In an extreme outbreak in Massachusetts, 1909, density of population no influence. Most cases occurred in old houses; direct contagion, with incubation period of one to fourteen days. A number of cases of indirect contagion by carriers; eleven out of 150 cases followed intimate contact with persons of old infantile paralysis—even of many years standing.

A large number of cases—half—give history of wading or swimming in sewage contaminated water. Recoveries equalled 10·8 per cent. of cases.

Diagnosis.—Fever, pains, paralysis—the earlier signs of cerebrospinal fluid.

Treatment.—Isolation of affected individuals. Disinfection by antiseptic sprays and douches of naso-pharynx in every person who may have been in contact with the affected family. Rooms disinfected with formalin. Internal administration of uro-tropin in view of the partial transudation of this substance into the cerebrospinal fluid in the form of formic aldehyde.

Immunity.—One attack usually confers immunity. Flexner and Lewis gradually increased doses hypodermically, until after four months the monkey was able to withstand a large dose of virus of maximum potency.

Monkeys which had recovered or survived from the paralysis were insusceptible to reinoculation, *i.e.*, they had developed an artificial immunity. Blood serum from such monkeys neutralised the virus in vitro. Serum and virus injected together failed to induce paralysis. Sero-inoculation, therefore, appears to be a practical means of prophylaxis. The blood serum of children who have survived an attack of poliomyelitis has been similarly employed. To be of use, serum must be employed early in this disease.

Neutralising serum cannot be obtained from the horse. The monkey appears to be the only animal in which the disease can be reproduced, and from which a protective serum can be obtained.

At the close of the discussion, it was proposed by Dr. C. S. Willis, seconded by Dr. J. S. Purdy:—" That it is the opinion of this meeting that a recommendation be made to General Congress that the notification of cases of infantile paralysis should be compulsory, and that the various State Governments should be advised of this recommendation."

This was carried unanimously.

I.

SECTION OF MEDICINE.

PRESIDENT'S ADDRESS.

The Address on Medicine delivered before the Ninth Session, Australasian Medical Congress, Sydney, 1911.

By W. T. Hayward, M.R.C.S. (Eng.), Adelaide, S.A.

It is my privilege to thank you, Sir, and the Committee of this Medical Congress, for the high honour you have conferred upon me in inviting me to deliver the Address in Medicine before this distinguished assembly of my confrères; an honour never to be forgotten, and ever appreciated. But great honours entail great responsibilities, and it was with no light heart that I accepted your invitation.

I propose to take as the subject of my address—

Some Phases in the Evolution of Treatment of Disease.

I make no apology for the apparent triteness of the theme. The Anglo-Saxon race is essentially a practical one, therefore, "Treatment" must appeal to every member of our profession. All our studies, whether anatomical, physiological, chemical, pathological, or medical and surgical, are devoted to the ultimate aim of curing disease or alleviating suffering. A review of the efforts that have been made towards this desideratum, should interest all of us.

The word "Evolution" is apt to conjure up in the mind a period of æons of time, but I hasten to reassure you that I am not going to inflict you with ancient history. I will not take you back to the times of Hippocrates, or even of Celsus; I will merely consider the evolution that has occurred since I first entered the medical profession, that is, in the early seventies.

I may remind you that the immediate period prior to 1870 is considered one of the most uneventful in the history of medicine; it might be described as a kind of "medical doldrums." The tempestuous times when our ancestors fought the battle of Venesection *versus* Stimulation had passed, and the recognised treatment that had held sway for an almost indefinite period had been absolutely annihilated. Reaction followed—almost torpor. The older practitioner still retained his belief in the value of drugs, and prescribed them more by rule of thumb than by reason. Symptoms were treated somewhat on the Irishman's rule, "If you see a head, hit it." Amongst the younger generation a spirit of "medical nihilism" had developed; the value of drugs was discounted, and "Expectant Treatment" came into vogue. I well remember a distinguished Edinburgh graduate telling me that all but twelve drugs might be deleted from the British Pharmacopœia.

This recalls to my mind an anecdote that was published in one of the medical papers many years ago. A distinguished foreign physician was being shown round the wards of a London hospital by one of its physicians; to the astonishment of his guide, he remarked on the attention directed to the "treatment" of the patients. When asked what was the procedure in the foreign hospitals, he replied, "We diagnose, and diagnose, and diagnose."

"What then?" asked his friend.

"Why, we make a post mortem examination, and verify the diagnosis."

I will not guarantee the absolute accuracy of the anecdote, but it exemplifies a phase of thought that was not unknown, even in Great Britain, in the early seventies of last century. The method may seem a bit one-sided, and, perhaps, hardly fair to the patient, but it contains the germ of all successful treatment: for correct diagnosis must be the basis of all our hopes of success.

I said that medical torpor was the characteristic of this period, but forces were at work that were destined soon to metamorphose medical science. The study of pathology was beginning to make itself felt. Pasteur was doing work that will render his name immortal, and in Scotland there was a star in the firmanent, hazy and obscured as it then appeared, that was destined before long to develop into the most glorious sun that ever blazed on medical science. Need I say that I refer to Lister, the man of whom it has been well said that he has saved more lives than Napoleon Bonaparte ever sacrificed. Perhaps some of you may say that Lister was a surgeon, and ask what has he to do with medical therapeutics—I answer that both directly and indirectly his work is responsible for the major share of the advances that have taken place during the last thirty years.

But we have made great steps in advance since the period I have mentioned. No one will dispute the statement that the treatment of disease has vastly improved during the past forty years. This is shown by the increase of longevity, and the diminution of the death-rate in all civilised countries. Of course, our friends the hygienists will claim, and fairly claim, that this result is due in a great measure to their efforts; but preventive treatment is a part of general treatment, and is not dissociated from it. I think, however, that we general practitioners can claim that we also have had a hand in the general improvement.

Professor Osler has enunciated five causes that have transformed medical work:—

I. The discovery of the cause of many of the great scourges of humanity.

II. Fuller knowledge of the etiology of disease, which has induced the practice of endeavouring to render the body unfavourable for the multiplication and action of disease germs.

III. The study of morbid anatomy, with clinical observation, which has taught us our limitations as to the treatment of incurable conditions.

IV. Study of the functions of organs and their perversion, which has led to the use of the products of metabolism.

V. The extended knowledge of bacteriology, which has caused us to endeavour to treat diseases of microbic origin by serum therapy. It is easy to illustrate by examples the correctness of these contentions. The glorious history of the students of tropical medicine teems with the triumphs obtained by the studies in the laboratory, and the self-sacrificing efforts of its votaries. One need only allude to their work in Cuba, Panama, and South Africa. We are fortunate in having in the Commonwealth a member of our profession, now occupying a high position in one of the States, whose work in this direction is recognised by all scientists. I allude to His Excellency Sir William Macgregor. I trust that his presence and assistance will stimulate the efforts of our Queensland friends in their endeavour to discover methods that may, if not exterminate, at least mitigate that Australian scourge—the mosquito. The treatment of our *bête noire*—tuberculosis—has been wholly changed of late years. Instead of combating the disease with drugs, we, by hygienic and other methods, endeavour to strengthen the resisting power of the body, and we are working carefully on the lines of vaccines, having recovered from the reaction that set in after the disastrous first experience in that direction. We must admit that, so far, our efforts have not been crowned with the success we should desire; but the future is hopeful; we are making progress; our death-rate is decreasing, and we are gradually learning what are our powers in the fight with the fell disease. Though I cannot for a moment agree with those optimists who talk about tuberculosis being stamped out in twenty-five years, I fully believe that the authorities of the next generation will have a very different tale to record from those of to-day.

The increased longevity of the race is greatly due to our better knowledge of the treatment required by elderly people, which knowledge has been obtained by the study of pathology. We do not attempt impossibilities; we do not try to cure the worn-out heart, or the almost wholly degenerated kidneys; we have learnt from the *post-mortem* room that individuals can live with hearts whose walls are almost as thin as paper or kidneys that are mere shells. We recognise that such organs cannot be improved by drugs, and that any attempt to stimulate them can only result in harm; so that when we recognise during life the possibility of these organs being in such a condition, our endeavour is to throw as little strain as possible on them, and to encourage the vicarious actions of other organs that may be more competent. Many an elderly patient, or a prematurely elderly one, may continue a life of modified usefulness, if the medical attendant is alive to his own limitations. A cognate subject is the recognition of conditions that may in themselves be abnormal or pathological, but are merely manifestations of the efforts of nature, to secure a pathological in place of a physiological balance.

What gains have resulted to Therapeutics, owing to the study of the functions of various organs, especially the knowledge obtained of the importance of the internal secretions? Who, twenty years ago, could have imagined the part played by the thyroid gland in the animal economy? And yet who, having seen the marvellous results of the administration of thyroid products, in a case of Myxœdema, has not been impressed with the potentialities of treatment when we shall have obtained a fuller knowledge of the whole subject ?

During the course of this Congress, we hope to learn a great deal about Vaccines and Serum Therapy. This subject is still in its infancy, but the results obtained so far are such that their potency cannot be gainsaid.

Dietetics have played an important part in the advance of therapeutics; more attention is now paid to what a patient may or may not eat; the tendency has been, I think, toward a greater liberality in the matter of diet, and the restrictions are, on the whole, fewer. As an example I would point to the dieting in cases of enteric fever. It is not so long ago since the only food allowed during the course of the disease, and for a fortnight after the temperature had fallen to normal, was simply milk, and it was astonishing the quantity that was consumed by the patient. Looking back to that time, I now recognise that many of the symptoms ascribed to the disease were simply due to the unassimilated milk, the most notable being the heavily furred tongue and abdominal distention. For many years past my practice has been not to insist on food being taken during the early stages of the disease, when anorexia is nearly always present, but to allow any reasonable amounts of water to be taken. As the appetite returns, I permit any easily assimilated food to be given, being guided as to quantity by the ability of the patient to digest it, and this independent of whether pyrexia is or is not present. Of course, I am speaking generally; exceptional cases will demand certain modifications. The general result of this practice has been that the patients have recovered from the disease with far less discomfort than was formerly the case, complications have been less frequent, relapses no more frequent, and the mortality has decreased. Then, again, in cases of renal disease and diabetes, I think I am right in saying that the restrictions in diet that were formerly considered absolutely essential are at the present day very much modified, to the comfort and welfare of the patient. After advocating a liberalisation of dieting, it may seem somewhat paradoxical to speak of the value of total abstinence from food for certain periods; but year by year I am more and more convinced that in this practice we have a valuable therapeutic agent. Unfortunately it is considered somewhat outside the pale of orthodox treatment, and moreover, it is difficult to persuade people that they can live without food for longer than twelve hours or so; but in certain cases no treatment is more effective. It is not surprising that this should be the case, for it can be justified on physiological grounds, and by common sense. Anorexia, as a symptom except in malignant disease, gives me little anxiety, for I find that if not interfered with, it sooner or later cures itself.

Perhaps the most notable change of opinion in medical treatment during the last twenty-five years has been in connection with the administration of alcohol in various diseases. Whereas formerly every case of typhoid, or pneumonia, or other acute disease, was treated with more or less alcohol, now it is generally recognised that it is not necessary except under certain definite conditions: it is not given as a matter of routine. Many physicians go so far as to say that it is never necessary or even beneficial. Speaking personally, I may say that though, in the early days of my practice, I followed the example of my teachers and used it extensively, yet year by year I gave less and less, and for some considerable time I have hardly ever employed it, and, to the best of my belief, my cases have done better without it than with it. I admit the possibility that I, and those who adopt similar abstention, may have gone to the opposite extreme, and for myself I keep an open mind on the subject. I feel strongly, however, as to the wrongness of medical men *ordering* stimulants of any kind, unless in their own minds they are *fully* convinced as to the necessity of so doing. To order a neurotic man or woman to take alcohol is almost as wrong as to supply them with morphia and hypodermic syringes.

Any address relating to the Advances in Treatment would be woefully incomplete if it omitted to pay tribute to our friends the surgeons. The intrusions of surgery into the domain of medicine have been fraught with untold benefit. Since the time when the lessons taught by the immortal Lister finally captured the surgical world, not a year has passed without some victory having been gained over conditions that had previously been considered hopeless, as far as medical treatment was concerned. It is needless to recapitulate the triumphs of the surgery of the abdomen, thorax, and head. We welcome them, and there is no sign of jealousy or envy in our congratulations. The accusation formerly levelled at us, that we delayed transferring our cases until surgery was of no avail, cannot now, I think, be charged against us. We fully recognise the value and necessity for surgical measures, and gladly avail ourselves of the surgeon's skill. But, greatly as I appreciate the services of the surgeon, I do trust that our surgical friends will restrain their ardour, and not consider that every pain in the right-iliac region indicates the necessity for appendectomy—nor trouble in the epigastrium, gastro enterostomy. Though I do so with bated breath, I feel bound to admit that, in my opinion, every case of appendicitis does not require appendectomy to be performed. I believe I am correct in saying that if the mortality tables of twenty-five years ago, when appendicitis was not a recognised disease, are compared with those of the present day, it will be found that the death-rate of the former, of all cases that could possibly be ascribed to appendicitis, is no greater than that of appendicitis in the latter. The deduction I draw from this is, that a very large percentage of cases of this disease recovered under medical treatment. Every medical man must recognise that in a certain number of cases appendectomy is the only possible method of treatment; the difficulty is to discriminate between these cases, and those which would recover under medical treatment. There is no doubt great force in the argument that if there *is* this difficulty, why run the risk of making a mistake that may be a fatal one: therefore operate in all cases. To my mind this is not a scientific argument: I think we should endeavour to *learn* to discriminate. In a very excellent address on "Some Latter-day Intrusions of the Surgeon on the Abdomen," delivered by Mr. Childs, of Portsmouth, published in a recent number of the *British Medical Journal*, he says:—"All must recognise, however, that, at best, surgery is a somewhat crude method of treating disease, and there are not wanting signs that many abdominal diseases, especially those of microbic origin, may in the not distant future, revert to the province of the physician, and be cured by methods less drastic and more scientific than the knife."

Earlier in the address he strongly advocated that cases of appendicitis should be operated upon before twenty-four hours had elapsed, from the initial symptoms. If this becomes the recognised routine treatment, and I think it bids fair to be, what possible chance has the physician to evolve "less drastic and more scientific methods"?

With the incursions of the surgeon into the realms of medicine, the development of serum therapy, vaccines, organo-therapy, the X-rays, massage, electricity, &c., it may well be asked, what place is left for the use of drugs? Are they being eliminated from medical treatment? Judging from the budgets of literature on the subject, daily received by us, I should say "No." The British "Pharmacopœia" contains somewhat under 1,000 different drugs or preparations. In that handy volume, Martindale, I find that there are (speaking roughly) over 7,000, and it would be difficult to calculate the numbers in addition, brought under our notice by the various English, American, and German firms of manufacturing chemists. It would thus seem that the demand for drug treatment has not decreased, despite the advance of other therapeutic means. The redundancy of drugs is due to the expansion of pharmacology, the endeavour to treat abnormal conditions in the body, elucidated by scientific observations, by drugs that have been shown by laboratory experiments to counteract similar conditions. Everyone will agree with the desirability of developing the scientific side of medicine. To make it an exact science is impossible—it will always remain more or less an art. To my mind there is a danger of looking too exclusively on science, and overlooking the advantages to be obtained from the art. Without wishing to discount the valuable knowledge gained by the pharmacologist of the laboratory, with his experiments on the lower animals, I would contrast the ephemeral reputation of many of his drugs with those of the pharmacologists of old, whose observations were made on the effects of drugs on the patients under their charge. Even though it be empirical to give opium, mercury, arsenic, or ipecacuanha, in certain cases, because years of experience had proved them to be beneficial in similar ones, may it not be equally empirical to give nitrites because they reduce arterial tension, unless we are fully seized with the import of increased arterial tension? As a matter of fact, who of us when suffering from any particular complaint, would not take any drug, whether we know its *modus operandi* or not, if we knew from experience that it had proved beneficial in a similar case to our own. I take this opportunity of expressing my surprise and regret that venesection is so rarely practised. Most writers on the subject express the opinion that it might with advantage be adopted more frequently than it is; nevertheless it is still neglected. In certain cases it is the most potent remedial means we possess, and nowadays it is not merely a rule-of-thumb procedure, but can be justified on physiological and pathological grounds. Personally, I not infrequently employ it, ofttimes with marked success, in cases of Bright's disease, morbus cordis, and in conditions where the right heart is distended, with consequent cyanosis.

While doing our utmost to improve the science of medicine, do not let us neglect the art. Year by year we are discovering the reason why such and such an empirical treatment is effective, and it is not too much to say that in many cases the empiricism of yesterday is the rational treatment of to-day.

Many of you, Gentlemen, will remember the thoughtful and illuminating Address on Medicine, delivered by my distinguished predecessor, Dr. Rennie, at the last Congress. Those of you who, like myself, were not present, have doubtless read it, and it must have afforded thought for us all. The subject—The Influence of Mind over Matter—is one that must often have interested and perplexed us. It has been brought prominently before us lately by the Report of the Committee appointed by the British Medical Association, to investigate the question of "Spiritual Healing." The report, *inter alia*, declares that there is abundant evidence of the efficacy of mental suggestion in the treatment of many disorders; that those disorders are mostly functional; that pain may be alleviated in cases of organic disease, and a certain amount of improvement take place. If this be so, and I think the majority of physicians and general practitioners will admit that it is so, we have in mental suggestion a therapeutic agent of great power. To a certain extent we exercise it daily, frequently unconsciously. Ought we not to endeavour to develop it, and be in a position to utilise it systematically in suitable cases? I see nothing irrational or astonishing in the idea that one can influence the body by means of the mind. Pleasure or shame can cause a dilatation of the capillaries of the face; anger can cause them to contract; fear will induce rhythmical contraction and relaxation of the muscles, also loss of control over the rectum and bladder, it will also produce violent action of the sweat glands. Here we have definite physical results, due to exercise of the mind, in the shape of emotions. The manifestation of these emotions can be induced by another individual. Is it beyond the bounds of possibility that other physical changes could be induced by other stimuli?

The impression made on my mind after reading Dr. Rennie's address, was the necessity of strengthening and increasing the personality of the medical practitioner. Though year by year we are extending our knowledge of organic disease, and thereby increasing our powers in treating it, yet there are still the functional disorders that will ever demand our attention, and these will increase and become more severe as time goes on, owing to the stress and strain of modern life. The difficulty of differentiating between functional and organic disease will increase, and this difficulty will be enhanced in those cases when the functional disturbances are grafted on to organic disease, as is usually the case. This will tax all the powers of the physician, and surgeon too, for the matter of that. There was something in the saying of an old writer that "Every physician should also be a metaphysician." But the trend of medical education is utterly opposed to this aspect. Physics takes the place of metaphysics, and the greater part of the education of the medical student is devoted to matters of infinite detail. This applies to anatomy, physiology, chemistry, physics, materia medica and, to a lesser extent, medicine. The student has to assimilate the various theories scattered so plentifully throughout these subjects:—theories that are here to-day and gone to-morrow. Later on in his course, his time is taken up by attendance on long courses of lectures on the various special subjects, and he is expected to be fully cognisant of many diseases and conditions that he will probably never meet with in the course of his life. To be able to pass his examinations, his life must be one continuous cram, and at the time of taking his degree he is tightly packed with a mass of facts and theories, so that, to use a simile, he is "unable to see the wood for the trees." Fortunately the student's power for forgetting is as great as that for assimilating, and by the time he has finished his year of residence in a hospital he has jettisoned a considerable part of his former knowledge, retaining most that is essential, and starts his career in practice with a mental balance that, but for his practical experience in the hospital, he could never have acquired. Unfortunately, not every student becomes a resident.

This is not my idea of the education that is calculated to furnish the type of medical practitioner best suited for everyday work; for we must remember that it is the *general* practitioner that should be our first care; let *him* be fit and sound, and there need be little fear as to the higher branches of the profession. I believe the best specialists are those men who have graduated from the ranks of general practice—men who are thereby able to take a broader view of disease than those whose main experience has been in one branch only. To attain this breadth of view it is necessary to know something of men and women, of their strength and their weakness, of their foibles and even of their sins. Yet students, at the most impressionable periods of their lives, if they hope to attain any position in the pass lists, must pass their lives cribbed, cabined, and confined to the laboratory, the dissecting room, the lecture room, and their studies. If I might use an analogy, I would say that it is not the theological student, well versed in patristic lore and Greek particles, who is the most helpful as a physician to diseased souls, but the man who knows the ways of life, the thoughts, motives and temptations of the men and women to whom he ministers. The medical practitioner has not only to deal with cases when the signs and symptoms are definite, and the treatment more or less obvious, but with a large number in which signs are indefinite, symptoms exaggerated or suppressed, and the treatment very far from obvious. He must not only be able to *recognise* these signs and symptoms, but to weigh them, not only in their co-relation to each other, but in their relationship to the individuality of the patient. The only way to obtain the knowledge of this individuality is through mixing freely with men and women.

I would greatly reduce the number of lectures that students are required to attend, reduce the size of the text-books, eliminate the recondite and contentious elements in the scientific subjects, and let the special subjects be treated only in so far as they are likely to be required by the ordinary general practitioner. Instead, however, of allowing a student to pass his examinations on a 50 or 60 percentage, I should insist on an 85 per cent. By this means we could be assured that the newly qualified graduate had a good knowledge of the essential parts of his profession, which he would probably remember for all time; instead of a mass of unassimilated information that would soon be more or less forgotten. He would have been able to spend more time in the Wards and Out-patients' departments of his hospital, and the dead-house, and thereby attain more practical knowledge. Moreover, he would have a sound basis on which he could graft any special line of work that appealed to him. It is not as if the man's education ceases as soon as he obtains his degree; the earnest one continues it throughout his life. The knowledge available for the student of medicine cannot be compressed into a five or six-years course.

Which is the better system: to give a man a sound and solid education, sufficiently serviceable to meet all the calls likely to be made upon him during his early professional life, a basis on which he could raise a superstructure gained by subsequent work and experience, or to endeavour to cram his mind with an extended knowledge of professional subjects, to the exclusion of any possibility of advance in general culture, or knowledge of human nature? Under which system is the man most likely to be able to maintain his position amongst his compeers, or be best able to fulfil his vacation as a healer of the sick?

I have wandered far away from my subject—I crave your indulgence. My object has been to draw attention to methods whereby we, and those who come after us, may attain, so far as medicine is concerned, the attributes of a good physician—these I saw defined by the late Bishop of Oxford, Dr. Paget, the son of *our* Paget, as—" Sometimes to cure, often to alleviate, and always to comfort."

A REVIEW OF THE PRESENT SITUATION AS REGARDS INFECTIOUS PROTOZOA.

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(*Abstract.*)

Just as some thirty years ago the bacteria obtruded themselves on medical attention, so now we are confronted by the Protozoa as another group of minute beings which play a part of no small magnitude in the causation of disease. The researches of more recent years have shown that protozoal diseases are not the affair only of our confrères who live in warm climates; the duty of acquaintance with them is cast upon us all. The subject is not simple; the literature is voluminous and often contentious, and a working acquaintance with it is only to be maintained by considerable and continuous effort. It seems to me, therefore, that I might perform a useful service if I can place before you such a survey of the matter as will serve to orientate the various items of current news concerning spirochætes, trypanosomes, sporozoa, and the like, and which busy clinicians find very little time to put in order for themselves. The realm of the infinitely little into which our subject leads us comprises a multitude of small beings which, resembling partly animals and partly plants, are not completely either the one or the other. Ernest Haeckel proposed to solve the difficulty by creating for these beings an additional kingdom under the name Protista. For the purposes of this paper the usual text-book divisions will serve as an adequate guide, and we may, therefore, consider the Protozoa as comprising the five sub-phylla: Mycetozoa, Sarcodina, Mastigophora, Ciliata, and Sporozoa. As regards the Mycetozoa, the general characters of the group were described, and special reference made to *Plasmodiophora brassicæ*. As regards the Sarcodina, various *Entamœba* were dealt with, and reference made to the hypothetical microbe of scarlet fever, the Negri bodies of rabies, and the Guarnieri bodies of smallpox and vaccinia. Under the *Mastigophora*, mention was made of the principal described species of Spirochætes and Trypanosomes, of the Leishman-Donovan bodies, and of Trichomonas and Lamblia, it being noted that so far the only trypanosomes found in Australia were those of Surra in imported camels, and *T. lewisi* in rats. In connection with the *Ciliata*, it was mentioned that a case of Balantidium colitis had been observed during the current year. The various groups of the *Sporozoa* were mentioned, and reference made to the occurrence of *Eimeria stiedæ* (*Coccidium oviforme*) in local rabbits, and of *Eimeria avium* in local turkeys, and attention was called to the discovery of various *Hæmosporidia* in Australian animals.

In describing the life histories of the various protozoa, stress was laid upon the fundamental importance of their reproductive processes as determining factors in the modes of dissemination of the diseases which they cause.

HYPERNEPHROMETA.

By Sydney Jamieson, M.B., C.M. (Eng.).

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General remarks on their frequency of occurrence, their position in classification, their peculiar diversity of histological structure, and the difficulties surrounding their diagnosis.

Brief description of three cases, with demonstration of the tumours.

Short account of their morbid anatomy; their clinical phenomena; their differential diagnosis; and of the principles underlying their treatment.

Of all tumours of the kidney, the hypernephrometa are by far the most important and the most interesting.

Their importance arises from the fact that they are now regarded as forming about 75 per cent. of the malignant tumours of the kidney occurring *in the adult*.

They are most interesting because of the diversity of their clinical manifestations, the curious absence of uniformity of structure, and the satisfactory results of treatment when operation is carried out at a fairly early stage of their existence.

I regret to say that my three cases do not illustrate this last feature in every instance.

I purposely intend to describe the morbid anatomy of these tumours first and the clinical manifestations afterwards, because many of the most important points in the latter are most readily explained by a knowledge of the peculiarities of the former.

Time will not permit me to go fully into the discussion of the place occupied by these growths in the scheme of classification of tumours. As such work belongs to another section of this Congress, suffice it to say that, prior to the researches of Grawitz in 1884, great confusion reigned in the classification of these tumour growths, and the views then enunciated by him as to their origin from foci of tissue in the kidney resembling that of the suprarenal body have, within the last few years, been contraverted by Stoerk who brings forward strong evidence in favour of their development from the structure of the kidney itself.

Until the peculiarities of the histological structure of these growths were pointed out by Grawitz they were frequently classified as adenocarcinomata, and an examination of some parts of the growth would most certainly warrant such a view.

A definite opinion cannot, with any pretence to accuracy, be formulated on these growths until portions of the growth have been examined from several parts of the tumour. It is, in fact, through the neglect of this precaution that so much difficulty has, in the past, surrounded the pathological investigation of these tumours.

Such growths have at various times been described as angiosarcoma, perithelioma, endothelioma, adenoma, adenocarcinoma, and adrenal carcinoma.

All this confusion, however, is now a thing of the past, as the characters of the hypernephrometa are well known to all competent morbid histologists.

This increase of our knowledge is in no small sense due to the low mortality now surrounding the operation of nephrectomy at the hands of the properly equipped modern surgeon.

Brief Account of Three Cases.

Case I.

O.F., æt. 57. Dealer. Admitted to S.H., 12th June, 1903. Died, 15th June, 1903. (P.M. 1,851.)

Complaint.—Pain in the left side of the abdomen, difficulty in urination, passage of bloody urine and hæmoptysis.

Duration.—Three weeks.

About three weeks ago, quite apart from any special discomfort, he began to notice that he was passing urine containing blood.

This has continued (with short remissions during which the urine is quite clear) ever since.

The passage of the urine was unaccompanied by pain. There was some frequency of micturition—he has to pass urine every three or four hours.

Occasionally the blood is passed in the form of quill-like clots.

The pain in the left side is, at times, very severe, and is always aggravated by locomotion, but rest always affords relief.

The pain is located in the left lumbar region, and is not radiating in character.

Family History.—No history of tubercle in the family, and nothing suggestive of gout or gravel.

Previous Health.—Good on the whole. Had gonorrhœa and a chancre thirty years ago.

On Examination.—Some tenderness on deep palpation over the left kidney. The urethra is patent along its under surface for a distance of ¾ inch from the tip of the organ.

Per rectum the prostate is found to be a little enlarged, but not particularly indurated or tender.

The urine, from the time of his admission till the day of his death, three days later, contained not a trace of blood. It was acid in reaction, sp. gr. 1020, pale straw coloured, and contained no abnormal constituent.

His temperature during his stay in hospital was normal throughout.

Generally speaking he showed signs of anæmia and general debility, and gradually sank, dying on the third day after his admission. For a couple of days prior to death he suffered from diarrhœa.

Result of Autopsy.

Thorax—cavities.—A few old adhesions were found at the apex of the right lung.

Both pleural surfaces were studded with reddish, pink, and dark red nodules of new growth.

Pericardial sac healthy.

Right lung.—Weight, 3 lb. 1½ oz. The organ was almost solid with small new growths varying in size from that of a pin's head to that of a marble. The growths were for the most part reddish in colour, some of the larger ones showing paler areas. In consistence they were firm. The lung tissue intervening was crepitant, but red and much congested.

Left lung.—Weight, 2 lb. ½ oz. Was in a similar condition to its fellow.

Heart.—Weight, 8¾ oz. Very little epicardial fat. Muscle pale but firm; otherwise normal. Slight atheroma at base of aorta.

Abdomen—peritoneum.—Healthy.

Liver.—Weight, 3 lb. 13 oz. Organ somewhat enlarged. Colour, pale. Consistence, firm. Several dark-red growths were seen projecting from the surface. Similar growths were also seen scattered throughout the substance of the organ. They varied in size from that of a pin to that of a marble.

A small cavernous angioma was also found beneath the capsule of Glisson on the upper surface of the right lobe.

Gall bladder and ducts healthy.

Stomach.—Healthy.

Intestines.—A small nodule of growth was found in the mucous coat of the ileum, with characters similar to those in lungs and liver. It showed ulceration on its free surface.

Right kidney.—Weight, 7½ oz. Organ large and pale. Its surface was studded with dark-red nodules. Capsule not adherent. On section, the cortex was pale and somewhat swollen.

Left kidney.—Weight, 1 lb. 8½ oz. Organ contained a large, irregular, partly solid and partly cystic mass. On section the mass was found to have replaced to a great extent both the kidney substance and its pelvis. The mass was pale yellow and caseous looking in its centre, but at its periphery it was more solid looking and of an ochre yellow colour. Some areas of the growth were reddish, others brownish in colour. Surrounding the main mass was a collection of cysts with firm fibrous walls communicating with one another, and with the ureter. This cystic area represented the remains of the kidney; no trace of normal kidney substance was seen.

Bladder and Ureters.—The left ureter was dilated, its diameter measuring from 1 in. to 1¼ in. Its walls were greatly thickened. The opening into the bladder was patent, and nothing was found to account for the dilatation. The right ureter was healthy.

Bladder.—Healthy. Some enlargement of the left lateral lobe of the prostate.

Spleen.—Weight, 5¼ oz. Soft in consistence, and of chocolate colour. No nodules.

Pancreas.—Showed a growth the size of a walnut in its body. The growth was firm and yellow in its centre, with a dark-red periphery.

The *Bronchial glands* were considerably enlarged, pigmented and soft in consistence.

The retroperitonial glands were enlarged and red.

Brain.—Showed no abnormality.

CASE II.

M.R., æt. 45 years. Was admitted to Sydney Hospital on the 10th June, 1903, and died there ten days later.

She complained of swelling of the legs and of the body generally and stated that her illness was of five months' duration.

Her face was very pale; the eyes were bright and showed œdema of the subconjunctival tissues. There was also a generalised œdema of the arms, legs, abdominal wall, and tissues over the sacrum.

The general appearance was similar to what one sees, as a rule, in cases of chronic parenchymatous nephritis.

The œdematous parts were solid to the feel, but pitted on deep pressure.

The breathing was somewhat embarrassed—a condition of partial orthopnœa.

She was obviously in a very weak and exhausted condition.

Circulatory system.—Apex beat in left nipple line. Marked epigastric pulsation. Right border of heart corresponded with right sternal border. At the apex the first sound was impure. A soft systolic murmur was audible in and confined to the tricuspid area.

Lungs.—Dulness at the right base behind, with diminished breath sounds and voice conduction. The chest wall was generally œdematous.

There was no headache, no failure of vision, no albuminuric retinitis, no evidence of uræmia, and no muscular twitchings.

A bed-sore about the size of a five-shilling piece was present over the sacrum.

The urine, which was scanty in amount, was acid, sp. gr. 1025. It contained no albumen or other abnormal constituents, beyond some brick-red urates.

Shortly after admission some Southey's tubes were placed in the legs, and from these 96 ounces of fluid drained away.

She complained much of tinnitus aurium, and as no albumen was found, it was judged to be due to anæmia.

An examination of the blood showed a grave secondary anæmia.

The treatment consisted in the use of hydrogogue purgatives. strychnine and digitalin.

No improvement whatever resulted from treatment, and she gradually sank and died, apparently of exhaustion, ten days after admission.

The temperature at no time rose above normal. Nothing could be made out within the cavity of the abdomen; this was largely due to the fact that the intestines were greatly distended with gas.

At no time during her stay in hospital did the urine contain albumen, casts, blood, or pus; nor was there any history of blood having been passed with the urine.

Autopsy.

Thorax—cavities.—A few old adhesions on the posterior aspect of the left lung. A pint of blood-stained fluid was present in the left pleural sac. The right pleural sac contained a similar amount of the same kind of fluid. Pericardial sac contained two drachms of clear yellow fluid.

Right lung.—1 lb. 14 oz. The organ was very œdematous. Its lower lobe was consolidated from hypostatic pneumonia.

Left lung.—1 lb. 3½ oz. Similar to its fellow, but the consolidation was less marked at the base.

Heart.—Weight, 11¾ oz. Right ventricle and auricle much dilated. Small deposit of fat on surface. The wall of the right ventricle was thin, pale, and flabby. The tricuspid orifice admitted four figures. The other valves were healthy and competent. The left ventricle was somewhat hypertrophied; aorta and coronary vessels healthy.

Abdomen—peritoneum.—Recent plastic adhesions between the upper surface of the liver and the diaphragm.

Liver.—5 lb. 13½ oz. Organ much enlarged; irregular in shape. It extended about 3 inches below the costal margin. A considerable number of pale yellowish growths of varying sizes up to that of a small tangerine orange presented on the surface. The larger ones showed umbilication of their surface. On section, growths of all sizes up to that of a large orange were found scattered throughout the organ. In consistence these growths were rather soft; in colour they were yellowish with a pink tinge running through it. The larger ones had small hæmorrhages into their substance.

Gall bladder and ducts healthy.

Stomach and bowels.—Healthy.

Kidneys—right.—Weight, 5½ oz. Showed nothing beyond some passive congestion. Attached to its upper pole in the position of the suprarenal body, was a large, somewhat kidney-shaped growth, about 9 inches long and 5 inches broad. It was rather soft and nodular on its surface, and was attached to the diaphragm and spine by strong, thick fibrous bands of connective tissue. It was firmly attached to the kidney capsule, but did not invade it. The suprarenal body was completely replaced by the mass.

On section, it had a loculated cystic appearance in part. This was due to some parts having disintegrated. In consistence it was soft, and for the greater part of a bright yellow colour, with, scattered through it, darker areas, some of a pinkish and others of a greenish hue.

The mass weighed 3 lb. 8 oz., and was entirely retroperitonial.

Brain and cord.—Healthy.

Remarks.—I have included this case in my record because it was apparently a growth of the suprarenal body, and yet had all the naked eye and microscopic peculiarities of an hypernephroma.

Case III.

A.H., æt. 45. Admitted to Sydney Hospital on 30th May, 1910, and died there on the 10th July, 1910.

Complaint.—Cough, and shortness of breath.

Duration.—Eight months.

History.—Eight months ago was upset out of a boat, and as a consequence was attacked by pleurisy. Ever since then had suffered on and off from cough and dyspnea. During this illness she was aspirated three times, 52, 20, and 10 oz. of fluid being removed at each tapping, respectively.

Family history.—Nothing suggestive of tubercular tendency,

Previous health.—Always good.

On examination.—Obviously suffering severely from dyspnea. Features cyanosed; finger tips dusky. Respiration, 32; pulse, 94.

Chest.—Marked inequality of movement on respiration, the right side expanding considerably less than the left; some diminution of V.F. on right side. On percussion the whole of the right side absolutely dull from apex to base. On auscultation some moist râles are heard at the right apex behind and also in front over the two upper interspaces.

The physical signs on the left side show no abnormality.

An X-ray picture showed extensive dulness over the greater portion of the right side, such as one often sees in thickened pleura.

The *heart* was displaced to the left, the left border being 7 inches from the middle line.

The liver dulness was displaced somewhat downwards.

Progress.—On the third day after admission the right chest was explored in two places, but nothing was found except a little *blood-stained* fluid.

The sputum was examined, but found to contain no tubercle bacilli.

June 5th.—½ milligram of old tuberculin was administered, but no reaction followed.

June 10th.—1 milligram of old tuberculin was injected; no reaction followed.

June 12th.—Breathing very distressed. 2 milligrams of old tuberculin given; no reaction. One-twelfth grain of heroin administered; this gave great relief.

June 13th.—Still very distressed. Some pain over the lower border of the liver with some dulness in both flanks.

June 21st.—Condition no better. Suffered last night from a severe attack of palpitation.

June 25th.—Attacks of dyspnosa becoming more severe and at shorter intervals.

June 29th.—Is being kept under the influence of morphia to relieve the distress. Free fluid present in the abdomen.

July 7th.—Appears worse. Is much more distressed. Pulse becoming weaker. Good deal of cough and expectoration. Urine, acid, 1030. No abnormal constituents found.

July 9th.—Very exhausted.

July 10th.—Exhaustion with increasing dyspnea. Died.

The *temperature* on the day after admission rose to 99·5° F., and on each of the two succeeding nights it rose to 99° F.; thereafter it remained normal throughout.

The *urine* at no time showed any abnormality.

There was no gastric disturbance throughout the course of the illness.

Autopsy.

Body much wasted.

Thorax—cavities.—The right pleural sac was completely obliterated, the sides of the pleura thickened and infiltrated by new growth. The pleura on the left side showed slight involvement along the line of the capillaries.

Lungs.—*Right* was greatly shrunken and collapsed. It was infiltrated by a huge mass of growth occupying the lower portion. Nodules of growth were also scattered throughout the organ, and some of these appeared to block the lumina of the larger bronchi. *Left* was not collapsed; small masses of growth found in the peribronchial tissue throughout the organ, and also in the sub-pleural lymph spaces.

Heart.—The pericardial sac was filled with straw-coloured serum. The whole thickness of the pericardium was infiltrated by new growth, especially at its lower parts, but also in the neighbourhood of the right auricle.

Abdomen.—The peritoneal surface of the diaphragm was infiltrated with growth similar to that described in the case of the right pleura.

The *liver* showed several small masses of new growth scattered throughout it.

The *Stomach, Intestines,* and *Pancreas* showed no abnormality.

Kidneys.—Right was to a large extent replaced by a partly solid and partly cystic growth. On section, this growth was of a lemon yellow colour towards its centre, with a more ochre yellow colour towards the periphery. Small red and brown areas were also scattered throughout the substance of the growth. In places this growth was undergoing necrobiotic changes, and it was by this process that the cystic areas were found. *Left* appeared normal.

Spleen not involved.

Uterus and *appendages* were not involved in the growth. The uterus was somewhat enlarged.

Brain.—Not examined.

Morbid Anatomy.

One of the most striking features of these growths is their tendency to show a more or less *definite encapsulation.*

The tumour in the early stages is well defined and sharply delimited from the surrounding tissues, and fairly distinct even from the kidney substance.

Another feature of these growths is that they tend *to grow out from the kidney,* leaving a large part of the kidney to all appearances normal.

The tumour appears as a smooth nodular or bossy mass projecting from the kidney surface. Externally, it is of a yellowish-white colour.

In consistence the growth varies from an elastic mass to a definite fluctuation, large cystic areas being not uncommon.

On section, the growth shows a striking lack of uniformity, it being cut up into irregular areas by slightly marked fibrous bands. The section is mostly yellowish-white in colour, but some parts are more translucent and greyish-white, while others are caseating.

The most characteristic feature of this section, however, is the presence of irregular patches and sometimes of considerable areas of bright yellow colour. These patches resemble in appearance a deep-seated gumma, but are not so tough.

There are also a few smaller areas of reddish or brown colour of more fleshy appearance. This process of caseation may result in cysts of various sizes, and sometimes the walls of these cysts are calcified.

Another striking feature is the early invasion of the renal vein by the growth. So it will be seen that the naked eye appearance of these growths are so characteristic that the true nature of the tumour may be ascertained in the great majority of cases with a great amount of certitude by a mere inspection of its surface and section.

To sum up the morbid anatomy, the most important characters are:—

(1) The encapsulation (apparent).
(2) The absence of diffuse infiltration, so that some part of the kidney recognisably projects from the growth.
(3) The multiformity of the sectional appearance.
(4) The caseas areas and cystic spaces.
(5) The bright yellow areas.
(6) The invasion, in some cases, of the renal vein.

The Clinical Phenomena.

These tumours exhibit great variety in their degree of malignancy and rate of growth. The growth may be so slow that years may elapse between the occurrence of the first symptom and the recognition of the kidney disease.

It looks as though an hypernephroma can exist for many years as a benign growth and then acquire malignant characters. In this respect it resembles closely carcinoma of the thyroid.

On the other hand the tumour may grow rapidly from the first and show widespread metastasis within a few months. Again, it may be many years before metastasis occurs, and then often enough the metastatic growth is single.

Symptoms.

The symptoms are very rarely characteristic, and often are most misleading and not even suggestive of the disease.

The general health does not, as a rule, suffer in any way till long after the tumour is readily recognisable.

One of the commonest symptoms is *fever*, and this is apt to suggest pyonephrosis.

In many cases urinary symptoms are entirely absent, but when present, hæmaturia and lumbar pain are the commonest.

The hæmaturia is often profuse and may be associated with injury. It may spontaneously disappear for long periods.

The lumbar pain is a continuous dull ache, which is not affected by rest or exercise.

Renal colic may follow hæmaturia from passage of clots.

Often other symptoms occur which suggest disease of neighbouring organs, and of these intestinal and gastric symptoms are most common. Epigastric pain, nausea, vomiting, and loss of appetite may occur.

When the growth involves the renal vein, it may spread along to the inferior vena cava, and we may get œdema of the legs and varicocele—these conditions, however, are somewhat rare.

Now, as to the tumour itself:—The tumour is in the loin, which it fills from behind. In the later stages, when it is enlarged, it may reach the anterior wall of the abdomen.

In front of the tumour lies the colon, which may be recognisable by percussion, but in some cases only after distension of the large intestine by air. The colon as a rule lies over the lower and inner angle of the mass. The tumour moves slightly in respiration. Except in the latest stages the tumour is not as a rule very large—it is firm, tense, and elastic, and sometimes obviously cystic—at times the mass is distinctly tender.

In its typical form it is rounded and but faintly bossy, but frequently it has somewhere in its outline one distinct boss—the kidney itself.

Occasionally aberrant clinical types are seen, thus the primary tumour may be absolutely symptomless, and the patient may present himself to his medical attendant with a secondary growth in some distant part of the body. Such secondary growth is usually single and most frequently is situated in a bone.

This peculiarity—its metastatic growths most frequently occurring in bone—is shared by these growths, with growths occurring in the thyroid, the prostate, and the breast.

The metastasis is also often solitary for a long time, and in this respect it accords with metastasis of carcinoma of the thyroid.

A malignant growth of a bone in an adult should never be operated on without the condition of the kidneys and thyroid being carefully inquired into.

Differential Diagnosis.

The conditions most apt to be mistaken for the condition are:—

(*a*) Hydronephrosis.
(*b*) Pyonephrosis.
(*c*) Enlargements of the spleen.
(*d*) Growths of the stomach.
(*e*) Growths of the colon.

(*a*) *Hydronephrosis.*—It resembles this condition in its distinctness of outline, its mobility, its elasticity, and often in its long duration. It does not, however, give the impression of a single uniform cyst.

(*b*) *Pyonephrosis.*—It resembles this condition in being associated with fever and in elasticity and tenderness, but it is more defined and more bossy in outline, much more movable, usually of longer duration, and is but rarely accompanied by pyuria.

(*c*) *Enlarged spleen* may as a rule be differentiated by the fact that it is superficial, has a sharp edge, and the splenic notch often decides the point, but in some cases of hypernephroma the kidney substance may project from the surface of the growth and resemble to some extent the splenic notch.

(*d*) *Growth of the stomach.*—The differentiation here is sometimes difficult, as the gastric symptoms that accompany hypernephroma may closely resemble those of gastric carcinoma. The differentiation may be established by gastric analysis.

(*e*) *Growth of the colon* is generally small, deep and fixed, and the resemblance between it and hypernephroma is as a rule but slight.

Principles underlying their Treatment.

So marked is the tendency of these growths (although the cases I have quoted do not bear this out) to produce single secondary growths, and so slow are they to reveal their malignant properties, that these growths, together with some similar thyroid growths, are amongst the very few instances in surgery where a primary growth and a systemic metastasis may be operated upon with a rational hope of obtaining a cure.

The clinical character of these growths, however, are so peculiar that failure to diagnose is most likely to occur.

Once the diagnosis is established the indications for treatment are clear. A kidney containing an hypernephroma *must* be removed. One may feel tempted from the apparent encapsulation and localisation of the growth to excise it, but such treatment will be of no permanent avail.

Although tumours of the colon often involve the kidney in their spread, hypernephromata rarely involve the colon.

In their removal there are four technical points that require attention:—

(1) The extent and connections of the growth and the condition of the other kidney must first be ascertained.
(2) Free access should be made, not only to the growth, but especially to its pedicle.
(3) The pedicle should be dealt with at an early stage.
(4) The perinephric fat should be freely removed along with the growth.

In order to carry out these procedures satisfactorily, a preliminary trans-peritoneal incision should be made so as to thoroughly lay bare the extent and connections of the growth and to inquire into the condition of the other kidney and abdominal organs.

Early access to and division of the pedicle is necessary so as to lessen shock and to lessen the risk of detachment of emboli of the growth during manipulation in those cases where the growth involves the vein.

These, then, are the most important matters to be attended to by the surgeon in his dealings with these growths, and it is remarkable how good the results are even in cases of extensive growths and in those where a single metastasis has occurred.

MATHEMATICS AND MEDICINE.

By Guy Griffiths, B.A., M.D. (Syd.).

Sir Dyce Duckworth has well remarked that medicine is not a science, but an art based on many sciences.

Even were it a science it would not necessarily follow that it could be treated mathematically. Sciences are either exact or descriptive, and it is the former only which are capable of strict mathematical treatment. Descriptive sciences are sometimes termed non-exact, and though Huxley, in one of his essays, objects to this adjective "non-exact," it is really useful in indicating unsuitability for strict investigation.

Medicine itself, then, cannot be treated mathematically. The old iatromechanical and iatromathematical systems of Borelli and others are hopeless, almost as hopeless as the Pythagorean system which would explain all natural phenomena on the basis of numerical relations.

Nevertheless, mathematical procedures are applicable to some of the sciences ancillary to medicine, to those in which we deal either with numbers of like objects, or with quantities which can be measured and expressed in terms of a unit.

1. The simplest of these procedures are those of mere *enumeration and ordinary arithmetic*.

   (i) I need only remind you of dosage, and its regulation according to weight or age (Young's rule). We are accustomed to regard matters of this sort as perfectly simple, yet many of us find dosage on the metric system too abstruse for us; much more so the regulation of progressive doses of toxins, such as tuberculin. I have suggested elsewhere a scheme by which such regulation may be achieved.

   (ii) Simple arithmetical procedures apply also in the case of heat and its expression in degrees Fahrenheit or Centigrade, and the conversion from one scale to the other.

   (iii) The enumeration of blood cells. In addition to the ordinary methods attempts have been made to attain greater exactness, and at the same time to simplify the calculation required; such attempts, mathematical in method, are those of Grunbaum, Turton, Gabbett, and Griffiths.

   (iv) I merely mention the work of Beddoe on the estimation of the values of foods according to calories.

   (v) Ratios and proportions are often extremely useful: they are extensively used in anthropometry, *e.g.*, the cephalic index. In other branches of medicine they are less commonly employed, but are sometimes of extreme value, *e.g.*, the pulse-respiration ratio, the differential leucocyte count, the corpuscular-colour index.

   (vi) Weber's or Fechner's law, "Sensation varies as the logarithm of the stimulus," is essential to an important part of physiology.

It is interesting to note that a somewhat similar law holds as regards the relation of result to effort, *e.g.*, in the increase of the speed of a train or steamboat. So, too, in modern medicine. In order to improve our results in arithmetic progression, we must elaborate our methods and increase our efforts in geometric progression. Small advances cost little effort, great advances cost great effort—effort great out of all proportion to the amount of the advance.

It is not sufficiently recognised, that the dissatisfaction of the profession with the terms of contract practice depends to a large extent on this disproportion between increase of effort and improvement of result. The modern practice of medicine has become so complex that the fees which were adequate a century ago, when there were no thermometers, no stethoscopes, no specula, almost no methods of physical examination (except mere inspection), are now ludicrously inadequate. This inadequacy is emphasised by the increased cost of medical education, and the rise in the price of other services, and in the cost of living.

2. I might describe as employed in sciences ancillary to medicine the whole subject of *statistics*. I merely mention vital statistics and Mendelism.

3. *Probability* is another of the mathematical procedures which has its application in the medical sciences; for example, in the estimation of the expectation of life, or of recovery, and in the comparison of results.

of treatment by such means as Poisson's rule, a particular case of Bernouilli's theorem. Similar analytical methods have recently been applied by Sir Ronald Ross to the calculation of the risk of infection with malaria, and the incidence of the disease in known districts.

While this work is of great value, we must beware lest excessive enthusiasm lead us astray. Herbert Spencer has pointed out that it is absurd to attempt to apply mathematical methods to inaccurate data; an awful example of the results of neglecting this advice is afforded by the recent work of Professor Pearson on the effects of parental alcoholism.

4. *Hydrostatics* and hydrodynamics have their application in ventilation; also in the propagation of the pulse wave and the blood stream (Dawson). Sahli and Jossué have attempted to calculate the work of the heart from observation of blood pressure, pulse rate, &c.

5. *Physical chemistry* has lately been employed in the explanation of immunity by Arrhenius and Madsen, who have applied the law of mass action to the problem of the neutralisation of toxin by antitoxin.

Another branch of physical chemistry, cryoscopy, has been called into service in measuring the adequacy of the renal function.

6. *Optics* are obviously all important in ophthalmometry. Usually the practitioner neglects the mathematical study of his subject, and, himself ignorant of its principles, relies on rules laid down by mathematicians.

The physician who understands something of the focal length of a concave mirror will find it much easier to use a reflecting head-mirror than will he who is altogether ignorant of optics.

7. The theory of *projection*, a purely mathematical subject, is the basis of all X-ray work; without it the estimation of the position of bodies which throw a dark shadow on the photographic plate is impossible.

8. With *graphic methods* of representation we are all so familiar in the case of temperature charts, that the merest mention suffices. How many of us fully avail ourselves of these methods in other cases—for example, in representing hæmoglobin values and degrees of oligocythæmia rubra?

9. *Designing of Instruments.*—The best results can be obtained from stethoscopes, ophthalmoscopes, obstetric forceps and other instruments, only when they are designed in accordance with mathematical principles; improvements are made only by those who bear these principles in mind. Think how much we owe to Helmholtz and his school!

Such then are a few of the uses of mathematics in the ancillary medical sciences. I have made no attempt at comprehensiveness. My remarks are intended to be suggestive only. So, too, are those which I now propose to make on the second division of the subject, *i.e.*, the value of mathematical ideas and principles in the explanation of phenomena which do not permit of accurate measurement, and so are not suitable for calculation.

Much of the reasoning contained in a text-book of physiology is of a mathematical type; throughout Harvey's "*De motu cordis et sanguinis*" one finds subject after subject approached in a mathematical fashion. The mere idea of circulation itself, the return to the starting point, is mathematical.

But it is not only in ways so simple and obvious as this that mathematical principles have their application in medicine. Consider the following:—

1. Euclid, in the 19th and 20th propositions of his sixth book, states that the areas of similar figures vary as the squares of their homologous sides; extending this to three dimensions we have the proposition that the area varies as the square and the volume as the cube of the linear dimension of similar figures.

   (i) At first sight it would appear that this theorem is useless in medicine, yet it affords us the explanation of the relation of growth, nutrition, and reproduction. The surface is the ultimate source of nutrition, and this proposition shows us that as the organism grows the volume increases more rapidly than does the surface, and only by division can the normal relation be restored. (Spencer.)

   (ii) Children are specially sensitive to the effects of exposure, partly indeed, because their skins are thinner than those of adults, but chiefly because, as the proposition asserts, a child has more surface per pound weight than has an adult, and so is liable to more rapid reduction of temperature.

   (iii) The convolutions of the brain, the valvulæ conniventes of the jejunum, and the folding of squamous carcinomata (Pearce Gould, B.M.J., 1910) are all explained by this same Euclidian principle.

2. The theory of *maxima and minima* explains not only the plan of construction of the bee's cell, but also the spherical form of unruptured hydatid cysts, and of sebaceous cysts, and the approximation to spherical form of the gravid uterus.

In the architecture of bones, the theory of maxima and minima explains the hollow cylindrical shape, and the disposition of compact tissue in pressure and tension lamellæ.

3. Those who remember the principle of *hydrostatics*, that the pressure of a fluid or gas acts equally in every direction, will have no difficulty in understanding how the tiny adductor of the vocal cords can "resist the whole of the powerful muscles of the chest wall," and maintain the closure of the glottis against a strong expiratory contraction.

Sir Douglas Powell has shown that, as considerations of hydrostatics necessarily indicate, the resilience of the thoracic wall is in favour of expansion, and there is no resistance of the wall to be overcome by muscular action in calm inspiration.

4. The *doctrine of continuity* explains how it is that diseases cannot be classified on the pigeon-hole method. No sharp line of demarcation can be drawn around any morbid entity. The right method of classification regards diseases as so many points scattered over a field, here discrete and there more closely aggregated; the foci of aggregation constitute the various disease types.

Neoplasms cannot be separated into two classes, malignant and benign; all are essentially similar, all malignant, the degree of malignancy varying continuously from very great to very little. (Cathcart.)

Continuity again explains why epidemics do not cease suddenly, but fade away. The virulence of micro-organisms varies continuously, not discretely. At the end of each epidemic, *e.g.*, of plague, scarlet fever, measles, there are a number of cases of infection by a virus so mild that definite disease can scarcely, if at all, be recognised.

Similarly, death is a gradual process: there is no sudden onset.

5. Bishop Berkeley was accustomed to make fun of the doctrine of infinitesimals of different degrees. These seldom occur in medicine, but we do deal with *finitesimals*, and we must always remember that the large is not infinite and the small is not zero, and often by no means negligible. The improbable is not impossible. The medical man is too ready to assume that because he has executed some procedure a large number of times, and no harm has resulted, that therefore no harm can ever result. Too many of us think that the risk of certain operations is nil. Even if the operation had been performed 100 consecutive times with success, that would be no proof that success was certain. There might be some small chance of misadventure, so small as to produce no appreciable effect in 100 cases, but, nevertheless, quite real. We do not forget that although we have crossed the street thousands of times without being killed by a passing motor car, we may yet venture once too often; we are, however, ready to assume that our own particular system of aseptic technique is perfect.

In the same way the finger-print expert assumes that his system of identification is infallible. Those who think mathematically will not fail to perceive its limitations.

A well-known application of probability is that emphasised by von Leube, that in diagnosis the common is always to be preferred to the rare: always provided that the common disease is capable of producing the manifestations observed; *i.e.*, the diagnosis is to be made according to the *a posteriori* probability, and not according to the *a priori* probability.

6. Some nosologists have wondered at the *constancy of certain rare diseases*, *e.g.*, splenic fever. Perhaps twenty cases occur per million deaths. It might be thought that sometimes no case would occur among a million, and sometimes fifty or sixty; yet there always seem to be about ten to thirty. The statistical doctrine of the permanence of small numbers, a doctrine capable of mathematical proof, shows that this constancy is just what should be expected. (Bowley.)

7. The *upper limit of a pleural effusion* is not level, it is curved like the letter S. The explanation of this is not quite clear, but it is no doubt related to the effect of capillarity in raising fluid between two plates inclined to another at an acute angle, so that the upper limit forms a rectangular hyperbola, an effect which can be calculated mathematically. Had the mathematician more complete data, he might in the same way be able to show that the upper limit of a pleural effusion must form an S line. (Ellis.)

Another illustration of the value of hydrostatical principles in medicine is afforded by *pleurésie blocquée.* In this by no means exceedingly rare condition (I myself have seen two cases) no fluid flows into the exploring syringe, although the needle be introduced into the fluid in the chest, and aspiration made in the usual way, although, too, the fluid be thin and the needle of sufficient calibre. The explanation is related to Powell's observation referred to above, that the resilience of the thoracic wall is in favour of expansion. Normally, there is a negative tension in the potential pleural cavity. When a large effusion is present there is a positive tension in the cavity, and so the fluid runs out on exploratory puncture. On the other hand, the tension of the effusion under certain circumstances, at present ill understood, may remain negative, and accordingly, an ordinary degree of aspiration is insufficient to cause the fluid to run out.

It was only by clear thinking in accordance with mathematical principles that the method of expansion of collapsed lung was conceived, and experiments devised to confirm the correctness of the imagined explanation.

8. Where the true explanation of a phenomenon is not known various conjectures are usually offered; if one only, it is received, however absurd. So soon, however, as the true explanation is discovered the absurdity must be rejected. Such is the case with dilatation of the heart, formerly absurdly ascribed to high tension produced by contraction of the heart wall, but now shown by James MacKenzie to be due to loss of muscular tone.

9. Lynn Thomas, Hurry Fenwick, and others have shown that the *interpretation of skiagrams* is beset with pitfalls. It should be obvious that it is hopeless without a knowledge of the mathematical theory of projection, at least until such time as the subject is codified, and adequate rules laid down by competent persons. Then, and then only, will the non-mathematical radiographer be safeguarded against the manifold errors, as is the oculist in ophthalmology, unless indeed, Davidson's stereoscopic method is developed to a degree of completeness which at present appears highly improbable, so that it can replace the ordinary procedure.

10. In recognising cupping of the optic disc, we employ the mathematical principle of *parallax*, a method which might find application in other directions also.

11. Our knowledge of the mechanism of labour, especially of the dilating force of the bag of membranes, and the mode of separation of the placenta in the third stage, is due to reasoning and investigation on mathematical lines.

Such, briefly outlined, are a few of the uses to which mathematics have been put in the aid of medicine. Many other phenomena, which may, perhaps, some day be elucidated by similar methods, yet remain unexplained, or only partially explained; as for example, the complete or incomplete collapse of the lung, which follows upon great or slight opening of the thorax.

A mathematical principle which might perhaps find application in medicine is that periodic phenomena may be effects of continuous actions; thus, as the transcendent genius of Newton showed mathematically, the periodic revolution of a planet in an ellipse round the sun is the result of the force of gravity acting continuously on the moving planet. Has this no possible use for us in suggesting an explanation of menstruation, cyclic vomiting, or periodic insanity?

As the study of medicine progresses, more and more facts become capable of explanation in terms of physics and chemistry; there is less and less need to call in "vital force," a something unknown to dynamics, as the *deus ex machinâ*. Our president expressed it well last night: "Medicine is passing from the traditional and empiric to the rational and scientific." In astronomy, the most highly developed of the sciences, mathematics explains how phenomena, which formerly were supposed to be due to the direct and continuous guidance of the finger of God, depend simply on physical laws; so, too, will it be with physiology and pathology; when our knowledge of them becomes more fundamental and when our analytical powers are more highly developed we shall see that in them as in other sciences, all the phenomena are governed by inviolable law.

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THE USE OF TUBERCULIN BY GENERAL PRACTITIONERS.

By Alfred Edward Perkins, M.A., M.B., C.M., D.S.O.

Forty-two thousand nine hundred and twelve persons, according to Government Statistician Trivett, have died of Tuberculosis in the State of New South Wales alone during the thirty-three years ending 1908. How many others were infected by those who died, and how much suffering those 42,000 deaths entailed among the rest of the population, who can tell?

Chas. H. Mayo, in his presidential address at the International Congress on Tuberculosis, 1908, says:—" As but 10 per cent. of the population die of it, we have a right to say that few serious diseases, not self limited, tend more naturally to ultimate recovery than does tuberculosis."

That the death-rate from this disease has diminished in a remarkable manner during recent years in almost all civilised countries, being for New South Wales in 1908 only 57 per cent. of the rate which prevailed in 1876, is an evidence of more enlightened and more hygienic conditions resulting from the enormous strides in sanitary science, and the far greater interest in those improved methods of life required by governing bodies, health associations, intelligent municipal corporations, and these bodies, together with the whole of the medical profession, form an army of no mean proportions in dealing with this ever present insidious enemy.

In the great war, the general practitioner must always form a large proportion of the advance guard. He it is who first sees the case. To his credit or discredit the patient either is treated at once in a reasonable and scientific manner, or goes away to hang on for weeks, perhaps for months, without even knowing the serious danger threatening, until incapacity for work or severity of cough or hæmorrhage bring him to a standstill, when it is, perhaps, now too late.

If the general practitioner fails to recognise the very early incipient tuberculosis, either from want of keenness, or from lack of the necessary aids to diagnosis (and it is acknowledged on all hands that early diagnosis is of supreme importance for any form of treatment to offer the best chances of success), then so much the worse for the patient.

No one understands the position of the patient like the general practitioner; the family history, the constitution, the means, the possibilities of infection, the peculiarities of the individual, are frequently known to him alone; and that the patient may reach the sanatorium early, if fortunately he be able to afford sanatorium treatment, rests often on the verdict of the family practitioner alone. Who is in the position of the general practitioner to watch the members of the family, contacts with the tuberculous patients, to examine them, to test for the earliest indications of commencing disease, and to prevent the spread of the dreaded disease to others?

Certain cases, after most careful physical examination, cannot be recognised as tuberculosis until after the lapse of time and the possible conversion of a closed into an open tuberculosis with the discharge of bacilli. The X-rays and the fluoroscope give valuable evidence, but many a case still remains calling for the application of a specific diagnostic test. This, fortunately, the general practitioner now has in tuberculin.

Bandelier and Roepke, in their "Tuberculin in Diagnosis and Treatment," say:—"In general practice, too, the time is past when the mention of tuberculin was taboo. If we aim at mastering tuberculosis, no matter by what means, it must in future become and remain the domain of the general practitioner. Most important of all, the tubercular patient, thanks to the specific diagnosis of the practitioner, would reach the sanatorium in good time, and in case he be obliged to leave again too soon would, thanks to specific after-treatment, be really cured."

Robert Koch, in 1890–1891, recognised, when he made his announcement that he had discovered in tuberculin a cure for tuberculosis, that he had also discovered a very powerful poison. His warnings and cautions were unheeded; tuberculin was administered to all sorts of cases, and many a poor, far-advanced consumptive was hurried to his grave. Ehrlich and others protested against this overdosing, and finally a storm of indignation arose, and reached its climax when Virchow, in 1891, stated he found softening and recent extension of disease in patients treated with tuberculin, and dying of tuberculosis, and the period of "tuberculin delirium" was over.

Since then an enormous amount of careful work has been done. The specified diagnosis and treatment of tuberculosis is a chapter accurately investigated and to a certain degree finally settled, and it remains for each of us to be guided in our use of tuberculin by the experience gained, and to carry on in the same careful manner the work so well originated by Trudeau, Goetsch, Petruschky, and others.

It must ever be borne in mind, when dealing with tuberculin, that we are using a powerful poison which we use in thousandths of milligrams instead of grains and drams. Tuberculin is, say Bandelier and Roepke, the most potent drug in the whole pharmacopœia. The greatest care is therefore necessary in dosage, both in diagnosis and in treatment. The only other requisite in diagnosis is a scrupulous surgical asepsis, whatever procedure we may follow, whether ophthalmo, cutaneous, or subcutaneous. Fortunately for the practitioner who has not the time to prepare his own dosage, the wholesale chemists supply sterile doses ready to hand.

There is no doubt that the fear of the poisonous properties of tuberculin, and the want of knowledge as to suitable dosage, for long prevented a more extensive use of it in diagnosis, but since 1890, innumerable tests have been carried out on cattle, where it has proved of even greater diagnostic value than in man. From 1895 to 1900, I carried out, following instructions of Board of Health, a small number of tests, and also many private tests, which demonstrated clearly that under certain conditions, tuberculin presented a valuable aid to diagnosis in the case of cattle. Following on the enormous amount of work done in other countries in the case of man, came the recognition of the fact that a positive reaction to the tuberculin test, if the dose be not too large, justifies the diagnosis of the existence of tuberculosis in the patient, but in case the patient fail to react, the disease may fairly be suspected of being non-tuberculous.

It is sometimes surprising to find that a patient presenting many of the physical signs of tuberculosis should, on testing, fail to react, and the test in this case proves the more valuable in that it saves one from recommending to a sanatorium a quite unsuitable case.

On the other hand, even while bearing in mind the suggestion of Treves that many tumours of the breast are tuberculous, Virchow taught that tuberculin injection caused a tendency to dissemination of the tuberculous process, and some hold even to-day such strong views that they would prohibit the use of tuberculin either for diagnosis or treatment, but the consensus of opinion has demonstrated that tuberculin used in proper doses, in properly selected cases, is free from harmful effects. Osler said: "An important point is its harmlessness. I remember no cases in which injurious effects have followed the injection."

Priestly Leech says: "In tuberculin we have a very certain and simple method of deciding whether or not a person is suffering from tuberculosis."

Lenhartz uses this pointed language in announcing the new tuberculin era:—"It is a defect in practice when tuberculin is not employed." M. Letulle, in his "Tuberculosis Curable" (1911), says: "We have a means of knowing if a tuberculous person is cured; it is sufficient to place a drop of tuberculin either in the eye or on a little scarification of the skin; under the influence of this toxic substance, the organism should not react."

Everyone who will take the trouble to learn a few simple instructions as to technique, who will observe a strict asepsis, who will guard against administering tuberculin to patients who are manifestly unsuitable, and who will exercise the greatest care in the performance of the work, will be amazed that he has so long allowed prejudice and ignorance to thwart him from the use of tuberculin.

The preparation that has been almost without exception used in diagnosis is the old tuberculin of Koch. Into the question of the preparation of this substance and its method of action in the tuberculous subject, there is no need to enter in this paper.

It is supplied in bottles of 1 c.cm., and keeps indefinitely if not grossly contaminated. It may be applied as a test of tuberculosis in one of several ways:—

Cutaneous Method—associated with the names of Von Pirquet and Schiek.

In 1903, V. Pirquet expressed the view that the capacity of the tubercular patient to react to tuberculin was conditioned by "reaction products of the nature of antibodies."

After much clinical work and much further experience, Von Pirquet announced the new modifications of this test in 1907.

The preparation used was Koch's old tuberculin. At first he employed a 25 per cent. dilution with ½ per cent. of carbolic acid. Since then he has preferred undiluted tuberculin, when only one single application is made. With children, the 25 per cent. dilution is preferable. The site chosen for inoculation is the inner side of forearm. The skin is cleansed, rubbed with ether, then with spirituous biniodide; two drops of tuberculin are placed on it about four inches apart. Scarifications are then made with a needle, first midway between the drops, then through the middle of the drops. It is not necessary to draw blood, though, according to Mills, "a drop of blood does not interfere with the reaction." Only sufficient pressure should be applied that a scab, however superficial, may be afterwards formed.

The point of the needle must be sterilised before each scarification.

After inoculation the tuberculin is allowed to soak in for some minutes, on a piece of cotton wool applied.

If possible, a daily inspection of the inoculation is made for the first two days, then again after three or four days.

A slight redness is visible in the immediate neighbourhood of the scarification after twenty-four hours and then disappears, leaving only a small brown scab on the natural skin during the next few days. After the scab falls off a pale scar is visible.

Inoculations with negative reaction give rise to same appearances as the control site between them. Von Pirquet recommends the beginner to regard reactions under 5 mm. in diameter as doubtful, and in such cases to repeat the inoculation.

The positive reaction has a latent period of from three hours to several days, but in most cases is well developed in eighteen to twenty-six hours, and is very distinct after an interval of two days. It is a slightly raised reddened patch, extending rapidly in height and extent; the average diameter is 10 mm., sometimes 20 mm.; the margin may be slightly swollen. As the exudation decreases, the redness fades and changes to violet, gradually fading and disappearing after some weeks.

McNeil accepts only a papule of a deep livid hue, which persists for not less than a week.

Von Pirquet warns against treating a patient for active tuberculosis on the strength of a reaction to his test. If the cutaneous inoculation is negative or doubtful it is repeated after some days. Of the specific nature of the cutaneous tuberculin reaction, occurring as it does only in the organism infected with tuberculosis, the whole literature of the subject leaves no doubt.

In certain diseases the specific reaction above detailed does not take place. Thus, in measles and in influenza, there is a complete disappearance of the process of reaction, which only makes its appearance again after about a week. During this period of anergy the tuberculous process extends rapidly, and the organism becomes at the same time peculiarly susceptible to attacks of other disease. The percutaneous method is that associated with the name of Moro, who holds that the application of a tuberculin ointment (5 c.c. of Koch's old tuberculin in 5 grams anhydrous lanolin) is a truly specific test for tuberculosis. The ointment, when stored in a refrigerator, remains potent for months.

A piece of ointment the size of a pea is rubbed into the skin of the abdominal wall for half a minute to a minute, and with moderate pressure of the finger. After inunction the part is left exposed for some time, and no dressing is necessary.

The reaction is indicated twenty-four to forty-eight hours after application by single isolated spots of reddening, analogous to the cutaneous reaction, and the reddening disappears after a few days.

In more severe reactions there is itching and twenty or thirty discrete papules, which gradually fade with slight peeling and pigmentation.

This method of testing is harmless, and is said to be effective. I have had but little experience of it. The symptoms which characterise a general reaction are in this case absent, and one cannot conclude that a method which lacks the important element, accuracy, is a method which should be implicitly relied on. Bandelier and Roepke tested this method in 100 adults in whom they had found bacilli, and in conjunction with the cutaneous and conjunctival method, and report the percutaneous method as the least frequently positive.

It may be applied in children rather than in adults, and though harmless, a negative result must not be taken to indicate conclusively the absence of tuberculosis. One can readily understand that the condition of the skin is an important factor in the satisfactory performance of this test, for it seems probable that less effect would be obtained in the case of a dry, hard, thick skin, or one containing a large amount of fatty matter. Hence it appears necessary in this test also to wash first with ether and spirituous biniodide.

The Conjunctival Test and ophthalmic reaction was introduced by Wolfeisner, and modified by Calmette. It is produced when in tuberculous subjects a 1 per cent. solution of dry tuberculin in sterile water is dropped into the conjunctival sac. In the normal individual this has no result, or at the best a slight reddening of the conjunctiva which quickly disappears; but in the tuberculous, six hours after the application, a marked congestion of the conjunctiva takes place, this reddening increases, and is accompanied by the usual signs of irritation. The greatest intensity of the reaction takes place in about ten hours; there is no pain, but sensation of swelling, and in twenty-four to thirty-six hours the reddening gradually disappears. There is no fever or any of the signs of a general reaction.

If in the first instance ·5 per cent. solution be used and prove negative, a second instillation of 1 per cent. is made in the other eye. There are said to be many contra indications to this test. Bandelier and Roepke suggest for the first instillation a 1 per cent. solution, and on repetition a 2 to 4 per cent. dilution. In children a ½ per cent. for first instillation, and a 1 per cent. solution for the next.

I have used the preparation put up by Messrs. Parke, Davis, & Co., and have found it very satisfactory. It is portable, is easily applied, and is of fairly constant composition. The patient sits with head thrown back, while the operator, standing in front, draws down the lower lid with the finger of one hand, and gently drops the solution from a glass pipette on the conjunctiva, allows it to remain bathing the conjunctiva for about a minute, and then allows the patient to close the eye.

This test is always open to the objection that an exact amount of toxic substance is not used, or if used is not absorbed; while on the other hand, in the event of corneal or conjunctival disease, an altogether superfluous amount may be absorbed, and consequently an altogether too great reaction obtained.

In the case of far advanced tuberculosis, this reaction does not take place.

Absolute contra indications are given by ocular disease, no matter of what kind and in what stage (Calmette, Bandelier, and Roepke).

It is reasonable that if the exact chemical nature and toxic power of a given quantity of tuberculin were known, and precisely that quantity would be absorbed, either by the skin in Von Pirquet's method, or by the conjunctiva in Calmette's method, we would have arrived at a stage when these tests might under all circumstances be warranted.

The diagnostic value of the conjunctival reaction receives almost universal recognition in France. After an exhaustive study of this method and a large number of tests, Calmette states that 92 per cent. of tuberculous persons react; while other observers, Baldwin, Wolff, Eisner, and others, place the reacting cases at 70 to 85 per cent.

They also conclude that, by observing the contra indications, this test is absolutely harmless to the healthy, non-tuberculous eye. It must be remembered, however, that in many cases where this test has been applied, treatment by subcutaneous injection is accompanied by a conjunctival reaction at each injection.

As a rule (Bandelier and Roepke), the indication for the conjunctival test is present only when in practice a therapeutic measure is dependent on its result, and definite considerations justify its preference to other diagnostic methods. In Australia, a country of long distances, it might prove of inestimable value to the country practitioner.

The Subcutaneous Tuberculin Test.—In applying this test the old tuberculin of Koch is most generally used. It is diluted with ·5 per cent. phenol, and two strengths only are required, 1 in 100, and 1 in 1,000. The solution should be clear, and may be heated to boiling immediately before use.

The patient's temperature should be taken every three or four hours for two or three days before the injection, and should not, according to Koch, attain a higher level than 99°. The temperature should be taken after the injection, exactly the same as preceding the injection; 8 and 11 a.m., 2, 5, and 8 p.m. are the times recommended for taking temperature, both before and after injection.

The temperature should be taken in the mouth, and the thermometer should be kept there for ten minutes. After the first injection the patient should be instructed in taking his own temperature, and care taken to see that he thoroughly understands instructions, as it frequently happens that they fail to shake the mercury down, and thus give a wrong reading.

Not long ago I received an urgent telegram—"Temperature 104°," which really was 100·4°. It appears a much more satisfactory proceeding to instruct some sensible person to take the patient's temperature, as the effect on a highly neurotic patient of reading his own temperature is frequently not beneficial.

The thermometer should be washed and disinfected both before and after taking the temperature. Taking the temperature twice daily is of no use whatever, as frequently the early morning temperature may be below normal, while the late evening temperature may also be but little above, whereas the temperature at 3 p.m. may be 101°, or even 102°.

The syringe should be all glass, and should be sterilised by boiling before each injection. The needle also should be sterilised before being used.

The skin is cleansed at the site of injection by rubbing with absolute alcohol (I generally use the spirituous biniodide, and without any untoward consequences), and after injection the puncture is sealed with a small piece of sterile rubber plaster—the simple technique of a cleanly hypodermic injection.

The site of injection usually recommended is the skin below the scapula; but since Dr. Camac Wilkinson demonstrated to me the technique, I have always given the injection under the skin of the forearm, and have never experienced any ill effects.

As to dosage, subcutaneously, authorities differ as to size. Koch recommended an initial dose of 1 milligram tuberculin, increased to 5, and finally 10 milligrammes. This dosage is now unanimously considered too large. Four injections should be made if no reaction take place before. Too great differences between successive doses are not to be made on account of the risk of severe reactions. The sequence of dosage recommended is then as follows:—Increase from $\frac{2}{10}$ milligram to 1 milligram for the second dose, to 5 milligrammes for the third, and finally to 10 milligrammes for the fourth and last (Bandelier and Roepke). In tuberculous patients, after an injection a reaction takes place. This consists in (1) slight swelling and redness, with feeling of stiffness, and tenderness on pressure in the immediate neighbourhood of the injection. This may remain for a day or two; then the redness gradually disappears, leaving a small area of thickening and hardness under the skin. In no case has the redness or swelling extended more than an inch from the site of puncture, and has always subsided in a day or two. (2) General symptoms, of which the most important is rise of temperature. Within eight hours after the injection the temperature begins to rise, reaches its maximum in ten to eighteen hours, and lasts for twenty-four to forty-eight hours. The smaller the injection, the later the rise in temperature, and it also reaches its maximum later.

In some cases the temperature does not rise for as much as twenty-four hours. This is called a late reaction. Hence the importance of taking the temperature after the injection at frequent regular intervals. A reaction temperature below 100·5° is considered slight, when it remains below 101·5° it is considered moderate, while above 102° it is called a severe reaction. The less the dose injected the less the rise in temperature. If only a small rise of temperature takes place after the injection, wait till the temperature again becomes normal, and inject again the same dose. This will probably cause an increased rise of temperature, or a cumulative reaction. A severe reaction begins with chilliness, or rigor, depression, sickness of stomach, or even vomiting, pain and tenderness in muscles of limbs, thirst, pains in joints resembling rheumatism, neuralgia, sometimes loss of appetite, with utter weariness and prostration. With the rise of temperature there is quickening of pulse rate, increase of cough, and also of discharge from sinuses, should such be present. The physical signs become much more definite, and any glands become more enlarged and tender on pressure. These signs, which occur about the foci of the tubercular process, afford valuable aid in diagnosis, and many a case may by these signs be removed from the list of unproven to that of tubercular. Many patients sleep much better as a result of the reaction, while some rest badly.

It is well to allow a patient to remain in bed the day after the injection, especially if the reaction be at all severe, but many go about without harm.

After the reaction has passed off, the patient improves in condition, feels better; I have never known of any patient who has not felt better after reaction from the injection of tuberculin, and some have had nearly a hundred injections.

There are certain contra indications to the use of tuberculin, but they are few and plain:—Firstly—Cardiac disease; secondly—Nephritis;

thirdly—Epilepsy; fourthly—Miliary tuberculosis; fifthly—Hysteria; sixthly—In cases with high temperature; seventhly—Menstruation.

In cases which are manifestly tuberculous the application of the tuberculin test is unnecessary, and Bonney points out that that resort to tuberculin diagnosis is hardly ever necessary, and many others hold that physical examination with the aid of X-rays and the microscope make this means of diagnosis hardly ever called for. But those of us who have been any length of time in considerable practice can call to mind many a case that has presented puzzling features, which would have been immediately cleared up by a diagnostic injection of tuberculin. Dr. F. Guy Griffiths mentions many such cases in his excellent "Studies in Tuberculosis," including some cases of my own.

It is recognised as axiomatic that the earlier the tuberculous patient comes for treatment the more certain becomes the possibility of restoration to sound health.

Koch exhorts practitioners, by all means at their disposal, especially with the help of examination of the sputum, and in doubtful cases by the diagnostic injection, to diagnose tuberculosis at the earliest possible moment (Bandelier and Roepke).

Quite recently a patient, æt. 31, presented herself with a tumour of the breast, over a year in duration; she had miscarried five months before, since which time the tumour had grown and become painful. It was hard, adherent to skin, nipple retracted, enlarged gland under the pectoralis major, neuralgic pains in chest round to side. Yet with Treves' insistence on the fact that many tumours of the breast are tuberculous, though unsuspected, ringing in my ears, I could not believe that it was not either simple inflammatory or carcinomatous until a pathological examination discovered its true nature.

How many contacts with members of a family who have died of tuberculosis, at the time apparently healthy, have afterwards developed tuberculosis it is hard to say; but I have in mind many such, and I am sure many of you who have long been in family practice know many such, too.

How easy would have been the diagnosis by a subcutaneous injection at the time, and many valuable months saved.

How many of us are there who would hesitate to attack a case of acute appendicitis, or an extra-uterine pregnancy, and yet how few attack a suspected tuberculosis; and yet in the latter the technical knowledge and manipulative dexterity is "as moonlight is to sunlight." Every general practitioner must, if the fight against tuberculosis is to be a success, become one of the advanced guard, find out the enemy at the earliest moment, before there are T.B. in the sputum, before the lungs are the seat of dulness and thickening, before the urine contains bacilli, before the case is an open one; or he must fight in the van against all those conditions which make for the spread of tuberculosis, as well as do his part against the actual enemy; or finally must fight in the rear guard, prepared to carry on the good work of the sanatorium, or the specialist when the patient may no longer be able to remain in the care of the specialist, and when he becomes sufficiently improved to be able to support himself.

It is necessary to remember that in testing children with tuberculin, the dose must be greatly reduced. Bandelier and Roepke recommend $\frac{1}{10}$, $\frac{2}{10}$, 2·5, and 5 milligrammes.

Many hold that the tuberculin test should not be applied after recent hæmoptysis, but in treatment, Dr. Camac Wilkinson specially indicates tuberculin for hæmorrhage. Powell and Hartley, "Disease of Lungs," state the tuberculin test should not be used in cases in which there has been recent hæmoptysis, owing to the increased local congestion which results. Bandelier and Roepke place recent hæmoptysis in the list of contra indications; the possibility of the occurrence of a hæmoptysis following on the rapidly rising dosage of diagnostic tuberculin must be admitted, though agreeing with Wilkinson that the case is quite different with the gradually increased therapeutic doses. There is a good deal of variation in the size of the diagnostic dose of tuberculin among those who work in this direction: H. Turban uses ·5 mgm., 2 mgm., 5 mgm.; Cornet uses 1 mgm., 3 mgm., 5 to 6 mgm.; L. Brown uses $\frac{1}{2}$ mgm., 1, 3, 5, and 8 mgm.; while some use $\frac{2}{10}$ at three-day intervals, and increase after if necessary.

As to the merits of the various methods of testing, L. Brown, in a report to the International Congress at Washington, says he believes the cutaneous method has not contra indications, but regards the conjunctival test as offering enough risk of severe injury to the eye to cause him to be opposed to the method. As to the subcutaneous, he has "used it in a large number of cases for eight years," and has yet to see, as a consequence, "anything more than a slight temporary untoward result." Powell and Hartley hold that the subcutaneous test should be reserved for afebrile cases in which the need for a certain diagnosis is urgent. The cutaneous test, if positive, is of minor value; if negative, it would point strongly against the tuberculous nature of the disease.

The conjunctival test is not, as a rule, to be recommended.

Of the other tests, the complement fixation test, the cobra venom test, the agglutination test, I cannot speak, having had no experience; but of the others I have used chiefly the subcutaneous test, because the subcutaneous test can do no harm and can only do good. Especially is this so when the test produces a focal reaction indicating in the case of the lung the exact site of the tubercular lesion. This focal reaction occurs in 40 per cent. of cases, and is represented by pain and an intensification of all physical signs. Cough is increased and sputum becomes more abundant, enabling T.B. to be found where previously none could be obtained.

In pregnancy, the value of the subcutaneous test is very great, as it establishes the diagnosis with certainty, and allows of measures being taken early in the interest of both mother and child and no bad result takes place as the result of the test. Dr. Guy Griffiths, speaking of the conjunctival test says,—"The conjunctival test of Wolff, Eisner, and Calmette, I rarely use; it is too dangerous." Four years ago Dr. Wilkinson began its use at the R.P.A.H., and thought it advisable to give it up.

There must be no misunderstanding in this matter of tuberculin diagnosis, no relaxation of the strictness of physical examination, assisted by the microscope and the stethoscope and X-rays, and no slumming of history. Dr. Guy Griffiths: "In one's enthusiasm for tuberculin as a diagnostic agent, one must not of course neglect the well-established methods of physical examination, which I thoroughly exploit in every instance." Bonney says that, "in diagnosis, tuberculin becomes necessary very rarely."

Powell and Hartley say: "The test is, however, not one to be used, save in those cases in which more light in diagnosis is, for special reasons, urgently needed."

L. Brown says: "No case of early or incipient pulmonary tuberculosis has been shown to fail to react to a dose of 10 mgm. or less of old tuberculin."

Turban, with his vast experience, says: "A pronounced reaction is never noted in the healthy."

That there is no necessity to make a tuberculin test in a case of well-marked tuberculosis of lungs, no one will deny, especially when the history, the clinical signs, the X-rays, and the microscopic examination of sputum says definitely, this patient is tuberculous; but it is just in those cases where more light is urgently needed that this test is most to be advocated. Just in such a case, that the patient may derive the best benefit from sanatorium treatment, or just here that a patient giving a history of tubercle, and presenting practically no physical signs, may be warned in time. Just here bacteriological diagnosis does not avail; X-rays give no shadow. Dr. Griffiths says—"When I addressed the members of the New South Wales branch of the British Medical Association on this subject, and urged the necessity of using a hypodermic injection of tuberculin, I was told that there was no need to labour the point, as all were prepared to admit its value and its use." I wish, therefore, to take this opportunity of pointing out that very few do use the tuberculin test, and many are still opposed to it. In this country, and the whole of the States of this Commonwealth, it happens that medical practitioners are separated by hundreds of miles from specialists, and it becomes the moral obligation of those so situated to make themselves masters of the knowledge and technical skill requisite for making a tuberculin diagnosis, so that then patients may not go on until they reach the third stage (Turban) before being submitted to sanatorium tuberculin treatment.

The establishment of dispensaries, which we look forward to with feelings of the highest appreciation, cannot relieve the general practitioner of the country districts from this duty of applying the tuberculin test in such cases as may be necessary, otherwise he will be neglecting one of the most valuable aids science has put into his hands.

Hitherto I have spoken of tuberculin only in relation to diagnosis; in treatment, tuberculin is equally necessary, and as the results of tuberculin treatment become more widely known, and the technique becomes better understood, a greater number of general practitioners will take the subject up, and so swell the band of those who make use of this method that before many years pass the great majority of general practitioners will use it daily. From Koch's own statements patients in the early stage of phthisis were entirely relieved of symptoms of disease in the space of four to six weeks, and from these observations he concluded that early phthisis was curable with certainty by means of tuberculin. Koch and many of his disciples recommend the carrying out of the tuberculin treatment in sanatoria; but a large percentage of those whom tuberculin can most benefit cannot afford the time or the money for a considerable stay at a sanatorium, and many who have families dependent on them can ill afford to be absent from their daily occupation.

Some insist that auto-inoculation from graduated physical exercise is sufficient without tuberculin administration, and of course for those who are not in employment graduated exercise in conjunction with tuberculin is advisable, but Dr. Griffiths has described many cases successfully treated with tuberculin "who never lost so much as one hour's work during the whole five or six months over which treatment extended."

Dr. Wilkinson has successfully treated many cases without the necessity of sanatorium life, and without taking them away from their occupations.

R. W. Philip, who has used tuberculin treatment since its inception, gives a decisive verdict in its favour. He says pronounced reactions are to be avoided, but mild reactions make for improvement; one of the most striking evidences of the good effect being a reduction of the symptoms of systemic intoxication, evidenced by an increasing sense of well-being on the part of the patient with which the changes in the lesions, where superficial and visible, correspond.

Everyday evidence is accumulating to show that the man who refuses to his tuberculous patient the beneficial effects of tuberculin is not doing his duty. L. Brown says: "The crucial point in tuberculin treatment is the selection of dose, and interval." The method of administration is far more important than the variety of tuberculin, and he who fails to consider that tuberculin is a powerful poison is a dangerous man.

The first dose of tuberculin should be so selected that all possibility of reaction is excluded.

Koch, Carl Spengler, and others have advocated that slight reactions under 100·4° F., are necessary for the best results.

The varieties of tuberculin most commonly used are:—Old Tuberculin; New Tuberculin, T.R.; Bacillary Emulsion, B.E.; Dimp.; Beranek, &c.

Each of the preparations of Koch are put up in bottles containing 1 c.c. or 5 c.c.

The old tuberculin has 50 per cent. glycerine, and the stock solution contains 1,000 mgm. in 1 c.c. Hence dilutions can easily be made fresh for each injection.

T.R. contains 20 per cent. glycerine, and in 1 c.c., 10 mgm. of solid residue.

B.E. is a suspension of one part of pulverised T.B. in 100 parts of distilled water to which equal parts of glycerine are added.

Stock solution contains 5 mgm. of solid residue in 1 c.c.

With old tuberculin the scheme of dosage should be as follows:—

$$\frac{1}{100}\quad \frac{3}{100}\quad \frac{6}{100}\quad \frac{1}{10}\quad \frac{2}{10}\quad \frac{3}{10}\quad \frac{5}{10}\quad \frac{7}{10}\quad 1,\ 1{\cdot}5,\ 2,\ 3,\ 5,\ 7,\ 10 \text{ mgm.}$$

These doses are to be employed in Turban I and II, where the general condition and bodily strength is good, and the mouth temperature during the day does not exceed 98·2/3°.

If a reaction occurs, the dose is not to be raised, but the same one repeated at a larger interval, or a smaller dose given.

With regard to the interval between successive doses, they may in general be given twice a week. In the case of tenths or hundredths, the interval may be one day. Above 10 milligrammes it should not be less than three clear days, and with larger doses of 100 mgm. and more, four to seven days.

New Tuberculin, T.R., contains immunising properties, and should be used in tuberculous cases which are neither far advanced nor suffering with high fever. Tuberculin has no effect on the mixed infections. But it is much easier to carry out a course of treatment with T.R. without severe reactions, and therefore without possibility of harming the patient, than with either of the other preparations of Koch.

The dose to begin with is $\frac{2}{1000}$ mgm., but if this cause a reaction, diminish the dose to $\frac{1}{10000}$. It is hardly ever necessary, however, to begin with so small a dose.

The reaction should not exceed half a degree in temperature, and must have subsided before proceeding to the next dose.

Generally the dose is doubled, up to 1 mgm., after 1 mgm. by 2 mgm., then by 3 mgm.

At first injections are given every two days, after ½ mg. every three days, above 5 mgm. once a week, and with higher doses even at longer intervals, up to 20 mgm., which may be repeated after intervals of a month.

By gradually increasing the dose, and watching carefully the pulse, temperature, weight, and general condition, it is possible to reach the maximum dose without causing one severe reaction, and this immunises against the products of the tubercle bacillus.

Many prefer this preparation in treatment. I have followed the directions given here in many cases with excellent results.

Bacillary Emulsion.—In early cases, or even Turban II with good bodily condition:—

Begin by injection of $\frac{1}{100}$ mgm.

Scheme—

$\frac{1}{1000}$ $\frac{2}{1000}$ $\frac{3}{1000}$ $\frac{5}{1000}$ $\frac{7}{1000}$ $\frac{10}{1000}$ $\frac{15}{1000}$ $\frac{2}{100}$ $\frac{3}{100}$ $\frac{5}{100}$ $\frac{7}{100}$ $\frac{10}{100}$ mgm.

at intervals of two days ;

$\frac{15}{100}$ $\frac{2}{10}$ $\frac{3}{10}$ $\frac{5}{10}$ $\frac{7}{10}$ $\frac{10}{10}$ mgm., at intervals of three days ;

$\frac{12}{10}$ $\frac{15}{10}$ 2, 2½, 3 mgm., at intervals of four days ;

4, 5, 6, 7, 8, 9, 10 mgm., at intervals of a week to 10 days.

This is considered by many to be the best of Koch's preparations. It is often given in very much reduced doses, and in any case it is well if reactions be marked to use smaller doses.

Bonney tabulates several series of cases treated with small injections, and in nearly every instance with marked improvement.

Fever due to uncomplicated T.B. is much more readily reduced by B.E., and Bonney is particularly emphatic on its use in the surgical forms of tuberculosis. Krause has used it in cases with the worst possible environment. In some cases fever disappeared which had withstood all other methods of treatment for a period of twelve months.

In the use of any of these preparations it is absolutely necessary to observe the most scrupulous asepsis.

To watch most carefully the pulse. Sudden increase in pulse-rate probably indicates an excessive intoxication; no injection should be given until the pulse-rate has subsided to normal.

The *temperature* is the most useful guide to the rate of increase, and spacing of dosage; as far as possible, keep the temperature as stationary as possible.

The weight should present a steady increase; any sudden loss of weight is an indication of excessive intoxication, and calls for the diminution of dose, or cessation.

To the man who understands what is meant by "surgical cleanliness," and to the man who is prepared to watch and note the effect of treatment, there is nothing of difficulty in tuberculin treatment.

To the man who has the requisite knowledge, and who has mastered the technique of tuberculin diagnosis and treatment, this paper is not directed; but to the general practitioner situated in country towns and villages, far from the specialist, and especially in those cases when the patient cannot afford to go to a specialist, I earnestly recommend tuberculin.

THE TREATMENT OF CONSUMPTIVES IN PRIVATE PRACTICE.

By Charles Bage, M.A., M.D. (Melb.).

The open-air life and constant medical supervision aimed at in sanatorium and hospital treatment are not always attainable by consumptives, and the family physician is frequently called upon to treat his cases under disadvantageous conditions. There is still a recognised field for his usefulness in making early diagnosis, persuading patients to submit themselves to treatment, advising them in accordance with what is attainable in each case, and instilling courage into their minds. The question has now arisen, to what extent bacteriological research has furnished resources that justify the treatment of consumptives in city or suburban surroundings.

My experience dates back to the period before the discovery of the tubercle bacillus, when the infectivity of tuberculosis was only surmised. Then, as now, over-crowding, debilitating influences, neglected catarrh, and irritation of the lungs by dust and grit were recognised as contributing factors, but the essential cause was thought to be inherited weakness. The onset of the disease was usually unperceived until physical signs had developed. In treatment, the value, in the early stages, of out-door life and liberal diet was generally conceded, but less attention was paid to rest than nowadays, except on the occurrence of hæmoptysis; sedative drugs and counter-irritants were mainly relied upon for the mitigation of cough. In many cases the progress of the disease was arrested, and in not a few instances recovery occurred, but there is no assured basis for statistical comparison with modern results.

The practical advantages that ensued upon the discovery of the germ are abundantly obvious in connection with prevention and diagnosis. In the domain of treatment we are still eagerly awaiting the discovery of successful processes for destroying the bacillus in the living subject, neutralising its operations, or procuring an absolute immunity from its effects. Until we are able to intervene effectively in one or other of these ways, we must continue to employ all the indirect means we possess for favourably modifying the course of the disease.

Apart from special methods suggested by bacteriological discoveries, the routine adopted in sanatoria may be regarded as the foundation of our best successes up to the present. In these institutions there is still much to be desired as to selection of cases and longer duration of stay; but there is no doubt that many lives have been saved by their agency, and they provide the whole community with conspicuous object lessons in hygiene. I refrain from quoting statistical information on this subject, as figures afford the least reliable criterion of the actual curative value of sanatorial and other methods of treatment. The initial conditions vary too much, and information as to the after progress of patients is too scanty, for fair statistical comparison. A striking feature of sanatorium treatment is its success in the elimination of the more tractable concomitant infections, which are so often accountable for a large share of the cough and fever of phthisis; and a new and highly theoretical aspect has been imparted to the regulation of exercise by the suggestion that the amount of exertion may be so graduated that the resulting auto-inoculation may have a maximum effect as an excitant of advantageous defensive reactions.

As bacteriological science advances, it presents problems of such increasing complexity that systems of treatment, based upon the numerous hypotheses advanced from time to time, are necessarily somewhat empirical in their character. In the present state of our knowledge, these methods, whether aimed at the destruction of the germ, the direct neutralisation of its effects, or the encouragement of the patient's natural powers of resistance, must be judged by their clinical results; bearing in mind, however, that clinical successes arising from the application of a theory do not necessarily prove its truth.

Numerous remedies of a purely germicidal character have been proposed, but for the most part only as auxiliary to the general treatment. Among recent suggestions are hypodermic injections of weak solutions of carbolic acid, intravenous medication with iodoform dissolved in ether, and the continuous inhalation of various antiseptics. More ambitious claims are made for the radio-active menthol-iodine treatment introduced by Dr. de Szendeffy of Buda-Pesth, but more specific information is required as to the material employed and the evidence of its remedial effects. It is to be remarked that any practicable drug treatment that would destroy the tubercle bacillus in the infected subject would at once make all other methods superfluous. The same remark might apply to the successful use of antitoxin from a foreign source, analogous to the familiar antitoxin of diphtheria. Attempts have been made in this direction, but without notable success, though it is stated in recent publications that anti-tuberculin can be demonstrated in the blood of tuberculous patients who have undergone treatment with doses of tuberculin.

Attention is now chiefly directed to the search for means of stimulating the special resisting powers of the patient to tuberculosis, and it is commonly assumed that these defensive reactions are directly or indirectly germicidal. The local and general disturbances following upon inoculation of a tuberculous subject with the various tuberculin preparations are now interpreted as being, simultaneously, evidences of a morbid sensitiveness to the poison of tuberculin, and manifestations of a defensive reaction, the former "anaphylactic" condition having been occasioned by the existence of a tuberculous lesion. It is difficult for one who is not engaged in the practical work of bacteriology to follow up the knowledge that is being acquired respecting the effects produced upon the blood and tissues by the introduction of foreign proteids. But it appears to be established that the toxin or the protoplasmic material of every microbe tends to excite the formation of a neutralising substance specific to itself in the blood and tissues of the infected subject.

When blood serum, modified in this way, is brought into contact with cultures of the original germ, the neutralising effects of these hypothetical substances are variously evidenced by the formation of precipitates, or the agglutination or solution of the germs, observable in vitro; they may display the opsonic quality of promoting the ingestion of their specific bacilli by leucocytes, or their antitoxic effects may be manifested clinically.

Theories of treatment based upon these rapidly accumulating observations are now daily being put to the test of experience. In Melbourne considerable use has been made of the new tuberculin, which is the triturated substance of the tubercle bacilli suspended in glycerine and water and then sterilised. My own earlier experiences of its employment were discouraging, for my patients exhibited uncomfortable reactions, without any compensating benefit that I could observe. Later on, when the opsonin theory was accepted, the amount of the dose was reduced and the intervals lengthened, and better results were obtained. But it is not possible in the course of general practice to make or obtain the observations necessary for regulating a course of treatment by opsonic measurements. The work demands special skill and uninterrupted time: the estimations are liable to considerable error, and are admittedly difficult to interpret with certainty; nor has the application of this method met with such success in the treatment of tuberculosis as in the case of some other infections.

The original tuberculin, so much valued as a diagnostic aid, may be assumed to be a solution of the toxin of the tubercle bacillus. It is a culture of the bacillus in glycerinated bouillon, evaporated to one-tenth of its bulk and filtered. As a curative agent it fell into disrepute through not fulfilling the enthusiastic hopes with which it was first received; but it is now taking its place with the new tuberculin as one of the recognised means of activating the protective processes of tuberculous subjects.

For the last two years I have used the filtered bouillon supplied by Professor Denys of Louvain. This variant of the old tuberculin is so extensively employed in Europe and so little known here that a short description of its constitution and mode of use will not be out of place. But I must first acknowledge my indebtedness to my old friend and fellow-student at the Melbourne University, Dr. W. W. MacGwire, who, after practising in New South Wales and Queensland, has spent several years in visiting famous European clinics, and has constantly supplied me with the most enlightening information and criticism. In addition to telling me of the estimation in which this treatment is held in European medical circles he sent me the Professor's publications, and encouraged me to make a trial of his methods.

The material used, known familiarly as B.F.D., is prepared by allowing virulent cultivations of the tubercle bacillus to macerate slowly in Koch's bouillon for long periods in closed vessels: some of the cultivations had been macerating for eight years at the date of my information. Another method employed with equal effect is to mix a bouillon that has been two years in contact with these bacilli and a bouillon that had been so treated for six months. In each case the preparation is filtered through porcelain, and the filtrate, without being heated or concentrated by evaporation, is bottled for use. What means of standardising it are employed I do not know, but its reaction-producing power is about equivalent to that of the old tuberculin.

On first using this preparation, Professor Denys began with a dilution of 1 part in 100, giving four ascending doses, proceeding similarly with a dilution of 1 in 10, and finally attaining a dose of 1 c.c. of the undiluted preparation. But in pursuance of his method, which was to avoid producing a reaction, he found it necessary to make a further series of dilutions, viz., 1 in 1,000, 1 in 10,000, and so on to 1 in 10,000,000. The diluting fluid employed is Koch's bouillon. Owing to his reluctance to alter the labelling of the dilutions he first employed, his nomenclature of these eight strengths is somewhat confusing, and upon making the successive dilutions for use upon my own patients, I found it convenient to number them from the weakest, No. 1, successively to the undiluted preparation, No. 8. The method of employment is to give regularly increasing doses twice a week beginning with the weakest dilution in febrile cases, and with the third or 1 in 100,000 dilution in afebrile cases. Four or five progressively increasing doses are given from each strength until 1 c.c. of the undiluted preparation is reached. But this regular increase of dose must not be practised in the event of a reaction occurring. Should a reaction occur the same dose must be repeated next time, but not until after the reaction has subsided; if another reaction occur, the dose is to be diminished, increasing again when tolerance is established; and even in the absence of reaction, it is recommended that at some periods the dose that has been attained should be repeated for two or three weeks, without increase. It is essential, therefore, to be on the watch for evidences of reaction. Sometimes these are found at the point of injection, in the form of induration, redness, heat, or pain, occurring some hours after the dose is administered; sometimes in the infected area, pain and oppression in the chest, increased cough and expectoration being suggestive of reaction in pulmonary cases. But the more common evidences are of a general character, such as headache, pains in the limbs, general discomfort, depression, weariness, restlessness, insomnia, loss of appetite, rise of temperature, and occasionally drowsiness. On several occasions I obtained marked reactions through impatiently advancing the dose when the quiet progress of the case made me suspect that the bouillon was inert. Great stress is laid by Professor Denys upon the observation of minute variations in temperature, and he recommends that the patient should be drilled into taking his temperature five times a day, keeping the thermometer under the tongue for seven minutes, or in the axilla for fifteen minutes.

The claims made for this treatment are that it immunises the tuberculous subject against his tuberculosis, and that the patient, after a second course of treatment a few months later will not react to the tuberculin test. A statistical diagram is published showing a large proportion of cures, especially in early cases, and it is said that even when the bacilli do not disappear from the sputum, they are often observed to have degenerated, becoming elongated and granular.

An attractive feature of this method is that it is not attended with distressing reactions, and that the patient's routine is so little disturbed by it. When I came to put it into practice two years ago, I was soon convinced that even in the smaller doses a steadying effect was produced upon the patient's temperature, and I then resolved to make a definite trial of its effects in the first instance upon advanced cases of consumption. Success in incipient cases would prove nothing, while harm might be done by keeping these patients in town instead of sending them away to better surroundings. Besides, I soon found that there is a limit to the number of consumptives that one can attend privately; most of them are impoverished by illness and would be unable to persist in a long course of treatment without special encouragement. My notes therefore relate to a comparatively small number of patients, for I made it a rule to advise removal from town wherever practicable in incipient cases. Two were cases of enlarged glands in the neck, these patients reacting positively to the Von Pirquet test. The remaining twelve exhibited the bacillus in their sputum; five of these twelve had cavities, and all but one had patches of consolidation: in four cases both lungs were affected.

Case I.—The most advanced case was that of a married woman aged 25, who had been in a sanatorium a few years ago. Ill four years, much wasted and very weak. Pulse, 120; respirations, 30; pain left side, cough, sputum contained bacilli, occasional hæmoptysis, occasional night-sweats. Deficient chest expansion both sides. Dulness on left side over whole of supra-clavicular, infra-clavicular, and infra-scapular regions. Cavity at left apex. Dulness and crepitation at right apex. This patient was laid up in bed under such open air conditions as could be obtained at her home in a dusty suburban street; her range of temperature was 97·6 to 102·4. The Denys treatment was begun on November 3rd, 1909, and after the first week the highest record was 99·6 until November 21st, when it rose to 100·4 for four days in succession, settling down practically to normal, 98 to 99·2, until the first week in January, when it rose to 100 during a spell of hot weather. On February 12th, 1910, after a small dose of the undiluted preparation, a marked reaction occurred, temperature rising to 103, and induration occurring at the site of the injection: it took six days for this reaction to subside, after which I resumed with a smaller dose, and worked up to a full dose on March 2nd, 1910. On May 31st, 1910, I began another course, working from half strength to full strength. Patient's temperature remained very steady, and by July 22nd she was able to get up regularly and walk a mile on level ground. On September 27th, 1910, X-ray examination showed the right side and upper part of the left side clear: the left base cast a dark shadow. Dr. Argyle, who made this examination, said that the shadow did not suggest pleural effusion, thus confirming the negative results of repeated punctures I had made in search of fluid. At this patient's earnest request I began another course on January 11th, 1911, and she is now again undergoing a series of weekly injections of the undiluted preparation. She is still very thin, but her appearance is much healthier than formerly. She can walk long distances on the level.

Her menstrual function has resumed, after being almost in abeyance for four years. There is some cough and occasional slight hæmoptysis, the latter usually supervening upon excessive exercise and exposure to dust. The sputum still contains bacilli. Her sleep is undisturbed, and appetite good. The chest expansion improved on both sides, but the consolidation is still evident at the left base. There is normal respiration at both apices. Pulse, 96; respirations, 25.

Instances like this occurred now and then in the old days before the bacillus was known. The ultra chronic state, which this case had entered upon, implies a fair resisting power, and one must allow too for the strong psychical influences of a new treatment. On the other hand, this patient has often assured me that she feels refreshed for two or three days after each dose: on two occasions I substituted a dose of simple bouillon to test her observation and found that she did not experience the usual benefit. After making all allowances, I have no hesitation in crediting the improvement in this case to the treatment by the Denys preparation.

Case II.—In contrast with the above is this case of a married woman aged 36. Ill six months, not greatly emaciated. I was called to her on the day of her discharge from a hospital, where she had gained weight, and her disease had apparently become quiescent. But she had travelled far, and in the evening her temperature rose to 104. Her right lung was broncho-pneumonic, and there were rhonchi all over the left side. This was on May 2nd, 1910. On the 11th the acute attack had subsided, and temperature ranged from normal to 101. A course of the Denys bouillon was begun on this date, and by the 18th the temperature ranged from 97 to 99. Respiratory movements seemed quite free, but there was a cavity at the right apex, and bacilli were found in the sputum. All went on quite uneventfully with the exception of one day when the temperature again rose to 104 with a good deal of bronchial disturbance. The temperature record showed a range of 97·4 to 99 for the month of June. But trouble began again after the course was over, indiscreet exercise and excitement bringing about a serious relapse, and death occurred on December 13, 1910, after six weeks of coughing and prostration, with very high temperatures. Shortly after she contracted phthisis, this patient had a severe septicæmia. This had been a large factor in her case, and it is no discredit to the treatment, considered as a means of evoking latent powers of resistance to tuberculosis, that it should have failed to effect a cure. This patient, like the preceding one, felt benefited by each dose.

Case III.—June 15th, 1910. A strongly-built, muscular man, aged 42 years. Three years ill with phthisis, and has spent three months at a sanatorium. High colour suggesting deficient aeration, venules showing conspicuously on right front of chest: fair expansion. Voice husky. Had night sweats regularly until nine months ago, occasionally since. Seven years ago had right pneumonia. Cough; bacilli in sputum. The entire right front was dull and breathless: no vesicular murmur audible in this region, but only distant creaking rhonchi. Loud pectoriloquy at the third and fourth ribs. The X-rays showed large shaded patches outside the heart shadow, diagnosed by Dr. Argyle as enlarged glands and a cavity, or else consolidation of the lung; the right apex was more opaque than left. In this case repeated courses of treatment have failed to relieve the laryngeal condition, the patient's voice being now merely a whisper. But apart from a few occasions of reaction and an intercurrent bronchial attack affecting the left side, the patient has held his own to an unexpected degree. The cyanosis has quite disappeared, and there is clear breathing at the apex of the right lung, and in the lower mammary region. The pectoriloquy is as pronounced as ever between these regions, with absolute dulness on percussion. The patient's muscles have softened and his pulse is more rapid; there is a large output of sputum containing bacilli. Yet considering the extent of his lesions, it appears to me that this patient has been saved from a rapid decline by the antipyretic effect of the injections he has received. The case seems to me to be peculiarly suitable for pioneer thoracic surgery, for there is a large cavity close to the ribs, surrounded by an old pneumonic consolidation, and almost certainly overlaid by an adherent pleura.

Thoracic surgery is a field awaiting occupation, and I am confident that before long it will be permissible to cut down upon tuberculous cavities in the lung and treat them surgically; it is difficult to calculate the effects of the resulting disturbances of intra-thoracic pressure, but it has already been shown that the artificial production of pneumothorax is a feasible means of procuring rest for a diseased lung.

The value of skiagraphy in the examination of consumptives has already been touched upon. On several occasions this diagnostic aid has enabled me to exclude phthisis, a full excursion of the diaphragm and a clear unmottled picture of the intercostal spaces negativing the existence of this condition.

Cases IV to XIV.—I do not propose to detail the remaining cases. But the adenitis cases improved after a short course, and I lost sight of them without again submitting them to a Von Pirquet test. One girl of 17 with cavities and both lungs affected, who had been discharged from a sanatorium unrelieved, seemed to derive no benefit; in another case a cavity healed up and cough has ceased. A healthy-looking young woman who had been in a sanatorium underwent a long course without any deviation of her temperature from the normal, though she coughed and expectorated tuberculous sputum at first; towards the end she had neither cough nor expectoration, but she used to exhibit occasional reactions evidenced by headache and malaise; she is the one who has no evidence of consolidation, and I have been unable to locate her lesion, though she has the wavy respiration suggestive of phthisis. The remaining six were definite cases of early phthisis, in each of which the area of consolidation cleared up in the course of twelve to sixteen weeks' treatment by the Denys method, supplemented by such hygienic advantages as could be obtained; two of them seem to have regained their health completely.

It would have been interesting and valuable to have compared this treatment with a similar use of the old and new tuberculin, and to have tested my patients by means of the various diagnostic reactions. But private medical practice cannot be carried out on the lines of scientific experimentation, and these observations must be left to a future occasion.

My conclusion, however, is that this method has a powerful immunising tendency, and that it is very much safer than any of the processes that are designed to evoke repeated reactions. Being regulated by clinical observation it is eminently applicable in private practice, and is an ideal method for tuberculin dispensaries, which are in my opinion much needed in Australia.

THE BACTERIO-THERAPY OF PULMONARY TUBERCULOSIS.

By Guy Griffiths, B.A., M.D.

To a pupil of Camac Wilkinson it is particularly gratifying to find at this Congress so much discussion of tuberculin, a subject on which a previous Congress (1901) refused to hear Dr. Wilkinson. His work is evidently not without effect.

The ideal treatment of pulmonary tuberculosis consists in a combination of the sanatorium and tuberculin methods—Koch (B.M.J., 1901—ii, 320), Wilkinson (p. 150), Pottenger (p. 205), Pel. (D.M.W., 1909, 1680). Fresh air, rest, dieting, tonics, graduated exercise, are always to be adopted where possible. However, it is often impossible, and in early cases especially they may be dispensed with and tuberculin employed alone. This method is essentially that of Dr. Wilkinson, who has shown that it is cheaper and more efficient than the sanatorium method, while it permits the patient to remain at work and does not break up the home by separating him from his family.

Varieties of Tuberculin.

There are enormous numbers of different tuberculins; the first is the T.K. or A.T.—the old tuberculin of Koch, "Koch's lymph"—and it is the best of all for testing, but is liable to produce violent reactions. T.R., a milder preparation, is extremely useful: it is not suitable for elderly persons, is perhaps less thorough in its action, and is extremely expensive. An excellent and much cheaper preparation which can be used as a substitute for it is B.E.* Alkaline Tuberculin (T.A.) and T.O. are liable to produce abscesses, and so are not used, T.O.A., the original old tuberculin, similar to T.K., but not concentrated by boiling, is useful as a preliminary for B.E. All these six are derived from bacilli of human origin. Corresponding preparations from bovine bacilli:—P.T.O., corresponding to T.O.A.; P.T. (Perlsucht Tuberculin), corresponding to T.K.; P.T.R., corresponding to T.R.; P.B.E., corresponding to B.E., are prepared from bovine bacilli.

Just before his death, Koch was at work on a new preparation, albumen-free tuberculin. It has since been further elaborated, and will soon be ready for use.

There are three rival methods of administering tuberculin (Wilkinson, Bandelier, Rolly, Emery, Allen, Pottenger). In each, one begins with small doses and progresses to larger ones. In the original method, that of Koch, elaborated by Wilkinson, this increase is carried on to enormous doses: 2,000, 3,000, 4,000, 10,000, or even 20,000, 40,000, 60,000 times as large as the original dose, and Wilkinson maintains that it is only by enormous doses like these that one can hope to produce permanent effects. Although it is stated that a repeated increase of this kind does harm by producing excessive reactions and by lowering the opsonic index, I have published evidence to show that reactions, and even occasionally large reactions, do good, and that the opsonic index is raised in spite of, or possibly even because of them.

In the second method, the mild method of Goetsch, one begins with small doses and increases them, but increases them very gradually only, and endeavours to avoid reactions. It is often useful, especially with timid patients, but is a method of half-hearted compromise, and has the usual failings of compromise.

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* *i.e.*, new tuberculin bacillary emulsion, sometimes called T.E.

The third method, Wright's method, consists of beginning with small doses and increasing them somewhat, but only very slowly and after intervals much longer than those recommended by Koch; the doses and the intervals being regulated in accordance with observations of the opsonic index, or with guesses as to the probable amount of the opsonic index (Allen). This method is still used, but many of those who were formerly most enthusiastically in favour of it have lost faith in it, and some of them have been completely converted to the Koch method. Emery (pp. 385–6) says:—"Admitting that the immunity is due to antibodies, these may be bactericidal substances or anti-endotoxins, and we have no reason whatever for thinking that the opsonic index affords any clue to the amounts of the substances formed. . . . And, of course, as is held by many, if the beneficial effects are due to repeated slight local reactions, acting in a way as yet not understood, the opsonic index is of little value." Again, he says of the Koch method: "the beneficial effects as recorded (as I have seen on a small scale) are undoubted."

Allen, formerly a devoted adherent, now admits: "It is a matter of doubt as to whether the opsonic index does really measure anything of value in immunity against the tubercle bacillus." (Edn. III—p. 214.)

The method of using tuberculin which I advocate is Wilkinson's method. As this is often applicable while the patients go about their usual work, it is sometimes called the ambulant method.

Professor Allen spoke yesterday of the value of tuberculin if properly used. It is just this proper use that is so difficult to learn. I know this from personal experience. Six years ago I gave up general practice to devote myself to this work, but, even after extensive reading and clinical study, I do not claim to know all about it.

The principle of the treatment is entirely different from that of any other therapeutic procedure, and so is difficult to understand, and still more difficult to apply; it requires elaborate and special study. The regulation of dosage is made on grounds quite different from what one would *a priori* think right.

At the beginning of a course of treatment patients with advanced disease are usually comparatively insensitive, and therefore small doses may be rapidly increased with little or no reaction and unfortunately with comparatively little good effect. On the other hand, patients in the early stages are usually extremely sensitive, even to minute doses, *e.g.*, ·001 c.c. P.T.O. If one repeats the same dose this supersensitiveness continues or even increases, and so one can make little or no progress. In order to get on with the treatment one must overcome this supersensitiveness by increasing the dose.

Scheme for Increasing the Doses.

The one usually recommended is similar to that of Bandelier and Roepke, which is of the nature of a modified arithmetical progression.

Lawrason Brown in Klebs gives a scheme somewhat similar to Bandelier's, and mentions also a logarithmic scale.

This latter is the ideal method and has been employed for years by Dr. Wilkinson. In it the doses are in geometrical progression, the amount of each being a multiple of the last. Where patients are sensitive this multiple is about 5/4ths or 4/3rds. Where they are less sensitive or where the original supersensitiveness has been overcome, this multiple may be about the square root of two. This sounds complicated, but is in practice very simple. Thus—if one dose be represented by 1, the second dose is rather more than 1·4 (*i.e.*, 1 multiplied by the square root of 2), and the third 2, the fourth 2·8, and so on, each dose being always twice the last but one. If one increases in this way the progress is smooth and regular without sudden alteration in proportion.

The length of time occupied by a course of tuberculin treatment, when one increases the dose each time in geometrical progression, is readily calculated. Thus, if each dose be one-third greater than the previous dose, *i.e.*, if it be four-thirds of the previous dose, we can find by the aid of an ordinary book of mathematical tables how many times greater the twentieth dose will be than the first, and how many doses it will take to arrive at a dose any particular number of times as great as the first. If the initial dose be ·005 c.c. P.T.O., and the last dose ·4 c.c. P.T., then the last dose will be about 4,000 times as strong at the first, because P.T. is about fifty times as strong as P.T.O. The logarithm of four-thirds is ·124 . . . . and the logarithm of 4,000 is 3·602 . . . . *i.e.*, nearly thirty times the logarithm of four-thirds, which shows that an increase of one-third on each of thirty successive occasions will be required to attain from an initial dose of ·005 c.c. P.T.O. to a dose of about ·4 c.c. P.T.

Of these thirty doses those up to ·04 c.c. P.T. may be given every third or fourth day, say, two a week—provided reactions do not interfere. The doses from ·04 c.c. P.T. to ·4 c.c. require a longer interval, five, six, or seven days, so that the thirty doses will require in all some sixteen or eighteen weeks, say, four months. Doses of 0·5, 0·6, 0·7, and 0·8 c.c. P.T., will require another four or five weeks, so that a full course of treatment from ·005 c.c. P.T.O., to 0·8 c.c. P.T., will extend, in the case of a patient who is having his first course of tuberculin, over five or six months. If for any reason, *e.g.*, special sensitiveness, the physician be compelled to begin with a smaller dose, or to interpose a longer interval between the doses, the course will be correspondingly extended.

If the patient be less sensitive, so that one may increase each time by the square root of 2, it will require about twenty-four doses to increase from ·005 c.c. P.T.O., to 0·4 c.c. P.T., for the logarithm of 2 is 0·301 . . . . which is one-twelfth of 3·602 . . . . (the logarithm of 4,000), and therefore the logarithm of the square root of 2 is one-twenty-fourth of the logarithm of 4,000. Thus about twenty-four doses will be required to proceed to 0·4 c.c. P.T., and about twenty-eight to 0·8 c.c. P.T. If no reactions interpose delay, one may administer such a course in four or five months.

Where the patient has had tuberculin before, and is being submitted to treatment for the second or third time, one may double the dose, or, possibly, even treble the dose, at intervals of two, three, or four days; and in this case the whole course may be completed in two or three months.

Since Koch and von Behring showed that cattle might be protected against injections of virulent bovine bacilli by treatment with weakened living human bacilli, Spengler suggested the use of P.T.O. for human beings infected with bacilli of the human type. Wilkinson has shown that P.T. is still more efficient for this purpose than P.T.O. Raw has offered the same suggestion, but so far as I know has not applied it. Where human beings are infected with bovine bacilli (tuberculosis of glands, &c.), they sometimes do better under T.R. than under P.T. Although these various tuberculins have individual properties to some extent peculiar to themselves, yet they all, both those of bovine and human sources, present a general similarity in the effect they produce—a group effect (Koch), (B.M.J., 1901, ii.—p. 214). Each one protects more completely against itself than against any other, yet each one protects to a certain extent against all others. Patients who have had a complete course of treatment with T.R., B.E., or P.T., invariably fail to react even to enormous doses of old T. After one gram of P.T. the patient may still react to 0·5 gram of old T., but not to 0·25 gram.

I have mentioned that reactions do good. Philip says "mild reactions do good," (B.M.J., 1909, ii.—261); also Emery, p. 386 (quoted above). The very extensive experience of Doctor Wilkinson shows that moderate reactions to 99·5, 100 or 100·5 should be aimed at; Loewenstein (D.M.W., 1910—1655) and Bandelier agree with this. Apart altogether from the good effect of slight reactions, patients frequently assert that severe reactions, 101, 102, 103, 104, 105, 106, although exhausting at the time, do still more good. Such reactions as this occur sometimes as the limit of toleration is overstepped, sometimes they occur by accident when the injection is made unintentionally into a vein. If this happens, the reaction follows much more rapidly than usual, perhaps within half an hour of the injection. As the tuberculin does not remain at the site of injection, but is rapidly carried into the general circulation, there is absolutely no local reaction (stich reaktion). Koch calculates that an intravenous dose produces five times the effect of the same dose administered subcutaneously.

Those who belittle the value of tuberculin and insist on the necessity for sanatorium treatment—not appreciating the enormous inconvenience and expense to which the patient with early tuberculosis is put when his doctor advises him to give up all work, probably losing his billet, for six months, in order to go into a sanatorium,—sometimes say that a similar effect can be produced by allowing the patient to inoculate himself with his own tuberculin from his own disease focus by graduated exercise. (Paterson, *Lancet*, Jan. 25, 1908, B.M.J., 1909, ii, 105.) As, however, other factors besides exercises, *e.g.*, excitement, a full meal, disturbed sleep, may produce auto-inoculation, it is usually impossible, in spite of the utmost care, to gauge the dose thus administered, while by the hypodermic method the exact dose may be administered, and increased in an exact proportion from time to time. It is possible that the final dose administered hypodermically is enormously greater than any that can be given by auto-inoculation. Graduated exercise should, of course, be employed in conjunction with the hypodermic administration, for those patients who are not at work. Where patients work all through their treatment it is out of the question.

I have no time to discuss the so-called autó-inoculation method of Paterson. I merely remark that so many other things cause a rise of temperature, even in health, *e.g.*, exercise, excitement, digestion, anxiety, mixed infection, that it is impossible to know whether or not auto-inoculation has taken place; secondly, that this method is inapplicable to patients who are well enough to go about their ordinary business, but who yet require treatment; and thirdly, that it is doubtful if by this method sufficiently large doses of tuberculin can be administered.

If the best method of administering tuberculin is by progressively increasing the doses, and if to repeat the same dose results only in the production of supersensitiveness; if again, as is the case, weak dilutions of tuberculin do not remain active but deteriorate, it is obvious that to employ trade dilutions whose strength is unknown (whatever may be marked on the container), is absurd and harmful.

I have purposely abstained from quoting case reports. I have published a large number, many of them of patients in the second stage, a few in the third stage.

When the disease was not too far advanced, the effect of treatment was usually good.

That others, who admit this, can deny that it is an argument in favour of tuberculin in early stages is to me extraordinary.

I have never seen a patient pass from the first stage to the second under tuberculin treatment. Very many of my patients followed their usual course of life during the treatment, and there was no remedial measure other than tuberculin injections adopted, therefore to these alone I ascribe the improvement.

The Limitations of Tuberculin Treatment.

It should be obvious that, as the tuberculin method of treatment aims at the production of active immunisation, it makes a demand upon the tissue cells of the patient, and so cannot produce wonderful results in the most advanced cases. All one can hope to do is to arrest the progress of the disease. Damage already done, *e.g.*, destruction of lung tissue, can never be repaired; healing, when it takes place, takes place by the growth of fibrous tissue, scar tissue, not new lung. In his original communication on tuberculin, Koch (D.M.W., 1890, No. 46), said:—

"Phthisical patients with large cavities, who almost always suffer from other complications, arising, for instance, from the penetration of other pus-forming micro-organisms into the cavities, or from incurable pathological changes in certain organs, &c., will probably only in exceptional cases derive any lasting benefit from the use of this remedy."

Dr. Wilkinson calls attention to these same limitations. He says (p. 23):—"There is, indeed, no royal road to success in the treatment of pulmonary tuberculosis, either by specific, rational, or other methods. Each case requires skill, judgment, experience, and in no small degree patience, and, after all, the best and most conscientious efforts may be baffled by the intervention of conditions that can be neither foreseen nor prevented." Again, he says (Wilkinson, "Tuberculin as a Specific Remedy for Pulmonary Tuberculosis," Aust. Med. Congress in Hobart): "The treatment of tuberculosis with tuberculin is specific. It is of no avail except for tuberculous diseases, and if other complicating conditions exist, if other micro-organisms are associated with tubercle bacilli in the morbid process, tuberculin may fail absolutely to modify the morbid process." Pottenger (p. 205) supports this; his actual words are: "These remedies will not remove fibroid tissue, nor will they restore tissue lost by ulceration. They cannot restore shrunken lungs, nor can they replace distorted or damaged hearts. They cannot destroy other bacteria which are the cause of mixed infection. They cannot furnish recuperative power when the system is totally undermined. In short, they cannot overcome the consumption which has taken place as a result of the tuberculous process."

In spite of thorough treatment, and apparent cure, relapses or re-infection may occur, especially in patients whose disease was advanced, unless care be exercised. The patients should be tested every six or twelve months with large doses of old tuberculin, and if reactions occur, subjected to a second, or even third or fourth course of treatment, otherwise their acquired immunity may disappear, especially after such an unfortunate accident as an inter-current illness, and the disease may appear afresh either in the old focus, or in some other part.

Attempts have been made to treat secondary infection by similar methods, *i.e.*, by the injection of killed suspensions of cultures of the bacteria obtained from the focus of disease. Unfortunately, these have been less successful, and it is doubtful if bacterins, other than tuberculin, are useful in pulmonary tuberculosis.

Restriction of Tuberculin Treatment to the hands of Specialists.

No definite rules can be laid down for the administration of tuberculin; although I have indicated a scheme by which the doses may be gauged in geometrical progression, yet the increase must be modified from time to time in accordance with the reaction and the condition of the patient. I consider it would be unwise for anyone to attempt to treat patients with tuberculin without having had special opportunities of studying the method. I myself assisted Dr. Wilkinson, and watched him at work for more than a year before I undertook to treat cases on my own responsibility. Many new remedies have been brought into disrepute by reckless use of them without regard to explicit directions laid down by those who introduced them. This was specially the case with tuberculin in the early '90's. "I desire earnestly to dissuade anyone from using the remedy in a conventional and indiscriminate way in all cases of tuberculosis."—Koch.

The use of bacterial preparations by those who are unversed in their properties is an extremely serious matter.

If the general practitioner does not know how to apply the tuberculin test, nor even examine sputum for T.B., how can he possibly be expected to undertake the therapeutic use of tuberculin?

Tuberculin, "the most active drug in the Pharmacopœia," should not be employed therapeutically except by those who have had special experience in the requisite methods, and have learnt to avoid most, if not all, of those mistakes which are, alas! so easily perpetrated. The art of using tuberculin is to be acquired only by the physician who combines the study of clinical pathology with the practice of medicine The mere laboratory worker is as dangerous in this field as the general practitioner.

Dr. Guy Griffiths, in reply, said, as regards the results of sanatorium treatment alone, he denied that they were permanent. They were good temporarily, but only temporarily. This sometimes applied also to tuberculin, but it was easy to repeat a course of tuberculin while patients continued to work as usual, but very difficult and expensive to repeat a long sojourn in a sanatorium.

Some of the speakers had said that reactions must be avoided; *a priori* it appeared likely that they were harmful; yet experience showed that they were not harmful, but beneficial, and that after them the patients improved and the opsonic index rose, even in spite of frequent reactions—perhaps because of the reactions; *a priori* arguments were valueless in the face of actual experience.

He had confined himself to pulmonary tuberculosis because that was the subject under discussion. In another section of the Congress he had spoken of localised surgical tuberculosis, which was much easier to treat with tuberculin than was pulmonary tuberculosis. The patients he had treated were not selected. He had treated patients in stage I without sending them to a sanatorium, because it was impossible to find sanatorium accommodation for them, and he believed that tuberculin alone was better than sanatorium alone. After Dr. Wilkinson, he supposed that he was the first medical man in New South Wales to treat private patients in a sanatorium.

Other speakers had disagreed as to the specificity of the subcutaneous test; he, himself, had no doubt on this. He had tested scores of healthy persons (relations of patients), and those with other diseases, and got no reaction. He deprecated the indiscriminate and reckless use of the remedy, especially by means of trade dilutions of unknown strength; for, whatever the original strength of dilution of tuberculin, deterioration took place in time. He preferred to follow Koch, the great master of the subject. He had studied Koch's methods under Dr. Wilkinson, and he was glad that Dr. Wilkinson's work, especially at the Royal Prince Alfred Hospital, had converted so many to a belief in the efficacy of tuberculin.

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A CONTRIBUTION TO THE HISTORY OF DISEASE IN AUSTRALIA.

By J. Burton Cleland, M.D. (Syd.).

For some years now, as time and opportunity gave occasion, I have been collecting information from old Australian Medical Journals and books on the early history of this Continent, as to the introduction and occurrence of various diseases in Australia, as well as items of interest in connection with various outbreaks. The present contribution consists of such data as I have been able to acquire referring to the three highly infectious diseases—measles, scarlet fever, and whooping-cough. I have endeavoured to obtain their first recorded appearances in Australia, and to obtain notes on the various epidemics, the descriptions of which have been summarised. Especially of interest is the mortality table, showing the different epidemic aspects of the three diseases. Thus, measles shows periods of marked epidemicity throughout Australia; scarlet fever shows the occurrence of epidemics often limited to individual States; and whooping-cough shows in general the constant presence of this disease, with occasional periods, reflected in all the States, of striking decrease in the mortality.

The occurrence in Australia of epidemics of measles and scarlet fever may be due to one of two causes: either the revival of the mouldering remnants of previous outbreaks, or a fresh introduction from outside. If the former is the more usual source, and if sea-quarantine could prevent fresh introductions, then, from a reference to the mortality tables, it seems possible to stamp out these two diseases in this continent by making them notifiable *during the absence of an epidemic*, and then isolating the patients and contacts. The deaths in some years from either of these diseases are remarkably low, and we may infer from this that the number of cases must also have been very small and well within limits that would enable such measures to be effective.

Most of the references to affected localities have been obtained from the *Australian Medical Journal* or *Australasian Medical Gazette*. When the name of the month appears in brackets, the date refers to the appearance of the note in one of these journals, the outbreak occurring at a varying period, perhaps a month, before this.

Measles.

According to W. C. Wentworth,[1] up to 1824 no cases of this disease had occurred in Australia.

Hirsch[2] states that measles reached the Hawaiian Islands in 1848, and the Australian continent in 1854, whence it was imported in the same year into Tasmania and New Zealand. His reference is to Hall (*Trans. Epid. Soc.*, 1865, ii, p. 70). Dr. E. S. Hall[3] records one death from measles in a child in the district of Hobarton, Tasmania, in 1853; 44 in 1854, and 2 in 1855. 27 of the deaths in 1854 occurred in the Orphan School there.

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[1] W. C. Wentworth. "A Statistical Account of the British Settlements in Australasia." Vol. II, 3rd Edit., 1824, pp. 92, 227.

[2] Hirsch. Handbook of Geographical and Hist. Path. Vol. I, 1883, p. 159.

[3] *Australian Medical Journal.* Vol. 2, 1857.

D. J. Thomas (*Australian Medical Journal*, 1867, p. 63) refers to the presence of measles and scarlet fever in Melbourne in the winter of 1851. This fact antedates Hirsch's statement of the introduction of measles to Australia in 1854.

In a discussion on scarlet fever before the Medical Society of Victoria, recorded in 1857,[1] references are made to this disease being preceded by measles in an epidemic two years before. Dr. Livingstone had seen both diseases co-existent on a voyage to Australia, which is significant of their introduction. Mr. Gilbee said the two diseases did not seem to require the same amount of attention as in England, the children often being seen running about.

We now come to a period when mortality returns of this disease are available for some of the Australian States. Reference to this table will show that from 1861 to the present date there have been nine considerable epidemics of measles in Australia, as evidenced by the deaths from this disease, and that in the intervening years between the epidemics, sporadic deaths had occurred in varying small numbers without a single exception in any year for the whole of Australia, although in some instances in some of the States there has been an absence of any fatalities.

An important point to notice in the table is that in the most isolated States, *e.g.*, Western Australia and Tasmania, the number of years showing blanks is much greater than in the case of the other States, but it must be remembered that their populations are considerably smaller.

These great Australian epidemics of measles may be grouped roughly into the following periods:—

1. 1860–2.
2. 1866–7.
3. 1874–5.
4. 1880–1.
5. 1884.
6. 1888
7. 1893–4.
8. 1898–9.
9. 1902.

The Epidemic of 1860–2.—Tratman (*Australasian Medical Gazette*, 1894, p. 179) mentions that, in this epidemic in 1860–1, in Perth 56 deaths occurred, of which 15 were children under 1, and 32 were between 1 and 5. He compares it with the epidemic there in 1883–4, when many adults were affected.

The Epidemic of 1866–7.—During this outbreak 1,057 lives were lost in Victoria, and 112 deaths were recorded from South Australia. I have not been able to obtain the number of deaths in any of the other States.

In a note in the *Australian Medical Journal* (Vol. ii, 1880, p. 73) it is said that this epidemic would seem to have first appeared in Western Australia, and thence reached the eastern continent. From information received from Mr. C. A. Patterson, of Perth, and recorded by Richard Helms in the "Anthropology of the Elder Exploring Expedition" (Royal Society of South Australia, Vol. xvl, part 2, p. 288) it appears that measles appeared in 1865 amongst the natives comprising the tribes inhabiting the coastal district of Western Australia from Geraldton to Albany, and those of the territories nearest adjoining these. The population was reduced by this disease to about half its former number.

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[1] *A. M. Journal.* Vol. II, 1857, p. 133.

D. J. Thomas (*Australian Medical Journal*, 1867, p. 83), in a very interesting paper on "The Recent Epidemic of Measles, Scarlet Fever, and Rubeola in Melbourne," describes a number of cases, to be more fully dealt with under scarlet fever, in which two of these exanthemata occurred in individual patients within a short time of each other.

"In 1868 some of the Pitcairn Islanders and their friends from Norfolk Island visited Sydney in the schooner "Pacific," and unfortunately contracted the disease of measles, which made its appearance amongst them when they were returning home, and obliged them to call in at Howe Island, where they remained aboard their own ship till they recovered; but the disease broke out on the island after their departure, leaving an unpleasant reminiscence of the visit." ("Official History of N.S.W." By Thomas Richards, 1883, p. 673.)

The Epidemic of 1874–5.—This epidemic is also said to have first appeared in Western Australia. Judging by the number of deaths, it seems to have been by far the most fatal recorded in Australia. 752 deaths occurred in New South Wales in 1875 from it, this being the first year of such returns in this State. From Victoria 1,797 are recorded, in South Australia 355, and in Tasmania 129.

The *Lancet* (19th June, 1875, p. 865), in a leader, thus refers to the outbreak, which, it says, during the last two years, had extended through the southern half of the globe. "In the latter part of 1873 it was introduced to Mauritius, probably from South Africa, where an isolated epidemic existed the year before. During the last quarter of 1873 and the first quarter of 1874, more than 2,000 deaths occurred . . . . In the summer of last year (*i.e.*, 1874) the disease gained a footing in South Australia, where a general epidemic had been unknown for nearly a generation. It spread rapidly amongst the population both of Adelaide and the surrounding districts. Adults suffered in large numbers. Later in the year it spread to Victoria and New South Wales, where, during the last hot season, its ravages have been severe, and an epidemic of scarlet fever accompanied it. From Australia it seems to have passed directly or indirectly to Fiji, where its results have been so terrible. At the latter place, 50,000 natives had died, death being often ushered in by dysentery."

The Epidemic of 1880–1.—This epidemic, which is said to have been introduced by passengers from the "Kent" (Carstairs, *Australian Medical Journal*, 1893, p. 348), seems to have been milder than the last, and not to have reached Western Australia. References occur to the disease being prevalent at Emerald and South Shore, Queensland, at the beginning of 1882; at Horsham, in Victoria, in the middle of that year; and in the Ipswich district, Queensland, about September.

It is interesting to note that during the years 1882–3 the following ships arrived in Australia with measles on board or during the voyage. They were:—"South Esk," which had 42 cases on the voyage, and which arrived at Moreton Bay in the middle of 1882; the "Orontes," with 23 cases, arriving in Sydney on August 19; and the immigrant ship "Roslyn Castle," which arrived in Sydney at the beginning of 1883.

In 1883, 20 cases were recorded from Perth towards the end of the year. Tratman (*Australasian Medical Gazette*, 1894, p. 179) mentions this epidemic, which especially affected adults. In 1883–4, there were 102 deaths, of which over 30 per cent. were of persons between 15 and 40, while there were 30 deaths of children under 1 year, and 22 of those between 1 and 5. Great numbers of aborigines were said to be affected. In September, 1883, measles and scarlet fever were present in Melbourne.

The Epidemic of 1884.—This epidemic seems to be associated with the previous one, of which, perhaps, it should be considered as part. It also seems to lead on to that of 1888. The following references in the *Australasian Medical Gazette* indicate presumably some of the chief districts affected:—Ravenswood, North Queensland (January); Morgan, South Australia, and Leven, Tasmania (March); Adelong (April); Wentworth and Hay (May); Dunolly, Victoria (June); Sandhurst and Eaglehawk, Victoria (October); Shepparton and Murchison districts in Victoria (December).

During 1884, the Adelaide Children's Hospital also suffered from the outbreak. In 1885, it was prevalent at Avoca, in Victoria, January to March; and in June, 200 cases in a fortnight were recorded at Camperdown, in Victoria.

In 1886 it was prevalent at Ballarat (January); and in 1887, 200 children at one Melbourne school are mentioned as having the disease (October); and it was prevalent at Sandhurst and parts of Melbourne and in Sydney in November; and during the same year in Adelaide. Dr. J. C. Verco records a small epidemic introduced by a boy from Victoria. 64 of the 78 deaths recorded in Victoria for this year came from Melbourne and suburbs.

Whether the epidemic of 1888 was directly attributable to the previous epidemic in Australia, or was introduced from overseas, cannot be ascertained, but it is important to note that towards the end of 1887 the following vessels reached the Queensland coast with measles on board:—The "Bulimba" and the "Duke of Devonshire," two immigrant vessels which were refused pratique at Thursday Island (November); and the R.M.S. "Dacca," which arrived at Cooktown on November 6, from London, and had 36 cases of measles between October 23 and November 21.

According to the *Australasian Medical Gazette* (May, 1888, p. 193) the outbreak of measles in Queensland was attributed to pratique being granted to the R.M.S. "Dacca," and to the fact that the immigration agent caught the disease through visiting a child on board. He then conveyed it to his wife and child, and thus it spread further. On the 15th May, 1888, it is said that measles were prevalent in every coastal town of Queensland, and almost epidemic in Brisbane.

In 1889 two mail steamers arrived at Adelaide with measles on board, viz., the R.M.S. "Oroya," on January 3, with 29 cases; and the R.M.S. "Orient," on April 11, with 15 cases.

The Epidemic of 1893–4.—There were 1,500 children affected with measles in Fitzroy, Melbourne, and 2,000 in Collingwood (July); and it was very prevalent at Ballarat, Bendigo, and Rosedale. There were said to be 60,000 cases in Victoria in August.

The deaths in Melbourne and suburbs are given as 2 for May of 1893; 42 for June, 186 for July, 116 for August, 31 for September, 3 for October, 2 for November. In this epidemic we find that 220 deaths occurred in

Sydney in September, and 140 in October. During the epidemic it is stated that measles were very prevalent at Tumut, New South Wales; Ingham, Queensland; Renmark, Minlaton, Normanville, and Port Macdonnel, South Australia, in December; at Rockhampton, Queensland; and Nairn, South Australia (January). A death was recorded in Melbourne in February, 1894. Twenty-one deaths occurred in Western Australia in 1893, where all ages were affected, and also aborigines.

J. G. Carstairs (*Australian Medical Journal*, Vol. xv, 1893, p. 438), in a paper on the then present outbreak of measles in Victoria, refers to seven epidemics in that State since 1853, as seen in the attached table. He considered these were not due to the revival of dying embers of previous outbreaks, but to fresh importations (by the "Kent," in 1880, and by the "Himalaya," in February of 1893). He gives some returns from 1880 to 1888, showing that there was a greater mortality in Melbourne, where the ships arrived, preceding that in the country. He gives some interesting information as to the introduction of measles into Geelong. Cases were introduced from Melbourne in October, 1887, and lasted for a year; no further cases appeared until March of 1893, when it was twice imported from Queenscliff, the first instance occurring through the medium of a boy who stayed there a week after the arrival of the "Himalaya." The type was mild; he describes some departures from the usual in the location of the rash, and mentions the intense infectivity before the eruption. The incubation period was 12–18 days; 21 cases ailed for 5 to 7 days before the eruption. About 900 cases were seen by medical men in Geelong, and probably quite 1,500 occurred—23 in adults. He mentions influenza with a rash in 1890–92, which was often mistaken for measles. In discussion on the paper, Dr. Rowan mentioned a case where 7 children in one family landed with measles, from which fully a dozen other cases arose.

Dr. Gresswell mentioned that, besides the metropolitan (Melbourne) district, the following places were affected by the epidemic, viz., Geelong, Queenscliff, Ararat, Ballarat, St. Arnaud, Bacchus Marsh, Lancefield, Portland, Port Fairy, Seymour, Bendigo, Romsey, Bollan, Colac, East Loddon, Bulu Bulu, Walhalla, Maldon, Wodonga, and others. He said that probably 20,000 children in Victoria had been attacked between February and June.

Of this epidemic in Sydney the return of the Board of Health contains the following statement:—

"13 deaths from measles occurred among the 311 cases admitted to hospital, being equal to a death-rate of 4·18 per cent.; the deaths registered in the metropolitan district from the same disease, excluding deaths occurring in hospital, were 388, and assuming that the rate of mortality of cases treated in their homes was the same as that of the cases treated in hospital, it is estimated that *there must have been some* 9,000 *to* 10,000 *persons attacked with measles in the metropolitan district during the epidemic in* 1893. Most probably the numbers were much larger, as only those cases which suffered from the disease in a more or less severe form were removed to hospital, and consequently it may be expected that the death-rate among such cases was higher than among those who were treated at their own homes."

The Epidemic of 1898–9.—This epidemic was separated from the previous one by a period of three years, in which the number of deaths varied from 2 to 7 in the various States. Measles became prevalent in Melbourne (April), Tasmania (June, continuing on to September) and in Sydney (September). In Dr. Borthwick's report of the health of Kensington and Norwood, suburbs of Adelaide, for 1898, he notices that the disease was increasing in amount since June, but probably would not be as severe as in 1893.

Dr. Scot-Skirving (*Australasian Medical Gazette*, Oct., 1898, p. 444) read notes, in Sydney, on this epidemic and that of influenza, which occurred at the same time. He considered the outbreak more severe than the two previous ones he had seen in Sydney. He had noticed rubella frequently, and scarlet fever and diphtheria each twice follow measles within three or four weeks. He had seen a proneness to an irregular pyrexia, reaching as high as 104° to 104·5°, after the rash of measles had gone—with this there was usually some glandular enlargement or sore throat. Several children had complained of an odd pain in the chest, behind the sternum, or else at the root of the lungs, for which he could find no cause, but suggested enlarged intra-thoracic glands. As regards German measles, he had seen this frequently follow true measles within a month; in some cases it was almost impossible to differentiate this from scarlet fever, but he mentions as important helps in this direction: pyrexia small, symptoms slight, seldom initial vomiting, throat rarely more than red, and the rash, though red and fairly diffused, showing a certain papular discreteness and never the intense red of scarlet fever. The lymphatic enlargement was also early and special, as opposed to its later appearance and correspondence to the throat condition in scarlet fever.

In discussion on this paper, Dr. Crago said he considered the epidemic more severe than previous ones. At the Benevolent Asylum, the mortality in children from broncho-pneumonia was high; as sequelæ, he had seen two or three cases of ulceration of the mouth and gums, one at first resembling cancrum oris, with later necrosis of part of the alveolus. Several pupil nurses were affected, and amongst these were cases of a return of rash, without any feeling of illness, within three or four weeks. He had also seen numbers of cases of rubella with a scarlet or morbilliform rash and enlarged glands at the backs of the ears and along the sterno-mastoid. Dr. L. F. Neill had seen many cases of second attacks in adults and children. The cases of measles he had seen had been mild. In the epidemic he had seen glandular enlargement precede the rash by several days. He had also seen enlarged axillary and inguinal glands. He thought that both measles and rubella were present, and that Dr. Crago's relapses were cases of the latter. He cited two patients who developed measles on the 18th and 28th August, respectively, and both had rubella on the 25th September. None of the patients he had treated for measles in 1893 had developed them in this epidemic. Dr. Kendall had lost two adults from convulsions after measles, one of whom had also pneumonia. Dr. Pockley remarked on the absence of corneal ulceration, and Dr. Morgan Martin mentioned having seen a few cases of second attacks of true measles.

Dr. Walter Spencer (*Australasian Medical Gazette*, July, 1899, p. 278) also wrote an account of the cases he had treated in this epidemic from May to October, 1898, in Sydney. These comprised 231 cases of measles, and 147 of rubella, most occurring in August. In the measles cases, he was impressed with instances of nocturnal delirium, obstinate vomiting and diarrhœa; in rubella, with enlargement of the sub-occipital glands, and an itching desquamation. Of 28 cases in adults, 7 had had it before; 29 second attacks occurred in children he had previously treated in 1893. He also says:—"An epidemic of a new description of skin disease in sucklings (which has been recorded elsewhere) was observed by me following the outbreak of measles and rubella. Two infants were born with it, though the mothers escaped." This disease was recorded by Dr. Spencer before the Royal Society of N.S.W., in May, 1899, as "A New Variety of Dermatitis Exfoliativa neonatorum."

In Victoria, Dr. R. L. McAdam (*Australasian Medical Gazette*, 1899, p. 3), in a Presidential address, refers to the epidemic of measles, still prevailing in remote portions of the State, as having been the cause, directly and indirectly, of many deaths.

In a discussion on the exanthema then present in Brisbane, Dr. Jackson (*Australasian Medical Gazette*, Oct., 1898, p. 447) mentions the presence of a mild type of measles. At the beginning of the epidemic, he had placed measles and rötheln mixed in a tent, yet there had been no contracting, in any case, of the other disease. Though scarlet fever cases had got measles, no measles cases had developed rötheln or *vice versâ*, and he therefore considered only one disease was present, which was either measles or rötheln. Dr. Marks said he had seen rötheln followed in ten days by black measles (a consultant had suggested scarlet fever). Several other speakers considered both these diseases were present. Dr. Connolly had seen petechiæ in measles in a woman; broncho-pneumonia had been rare.

Some account of the epidemic in Melbourne in the last half of 1907, and the beginning of 1908, is given by S. W. Patterson in the *Intercolonial Medical Journal* (1908, p. 258), based on 598 cases seen in the out-patients' department of the Children's Hospital. In these, the rash appeared on the first to sixth day, usually on the third or fourth. In one case, it was hæmorrhagic on the trunk and upper arms. In two cases there were prodromal rashes—in one, scarlatiniform, in the other papular erythematous—both followed next day by the true rash. The stage of invasion in 42 out of 175 cases was accompanied by vomiting. Severe epistaxis took place in two cases, on the first and second days, respectively, followed by the rash on the third day. Koplik's spots were noted in many cases, with more or less redness of the mucous membrane, sometimes almost a stomatitis. There was bronchitis alone in 26 cases, and with broncho-pneumonia in 43, of which 6 were fatal. There was croup in 10, which was diphtheritic in 3; there was tonsillar diphtheria in 1 case, and "coccal" tonsilitis in 2. Tuberculosis occurred in 2 patients; 4 cases of tubercular meningitis and 1 of tuberculosis of the spine occurred in cases who had had measles recently. There were enlarged post-nasal growths and tonsils in 28, earache in 13, otorrhœa in 10, severe stomatitis in 9, severe conjunctivitis in 4, severe vomiting during the attack in 40, convulsions during the attack in 2, and in 3 cases a generalised pustulosis of the skin as a sequela. General debility also commonly occurred after the attack.

Scarlet Fever.

E. S. Hall (*Australian Medical Journal*, Vol. iii, p. 85), in an article entitled "On the Medical Topography and Vital Statistics of the City of Hobarton, Tasmania, and its Southern Sub-districts for 1855," mentions that 18 deaths occurred from scarlet fever in the Queen's Orphan School, Tasmania, in 1843. This seems to be the first reference to the disease in Australia. Hirst ("Hand. of Hist. and Geogr. Path.," Vol. i, p. 176), quoting a paper by Hall in the *Trans. Epid. Soc.* (1865, ii, p. 72), attributes its first appearance in Australia and Polynesia to the year 1848, when it broke out almost simultaneously in Tahiti, New Zealand, and Tasmania, "but everywhere to a limited extent and in a very mild form." Speaking of a few years later, Hall, in his first-mentioned paper says:—"In the great epidemic of 1853—long to be remembered by almost every family in Hobarton—230, out of the 994 total deaths of the year, were caused by this disease. Many of them were adults. In 1853 the Orphan School was more fortunate than in 1843, it having escaped the visitation." He mentions, in this epidemic, the death of the wife of an officer and her child. He says there were 2 deaths from scarlet fever in infants under 3 years in 1851, 11 in 1852, 93 in 1853, 5 in 1854, and 1 in 1855. In 1858, it was again rife in the Orphan School, with one death. D. J. Thomas, in a paper to be noticed later on, refers to the presence of scarlet fever in Melbourne in 1851. James Motherwell (*Australian Medical Journal*, Vol. i, 1856, p. 42) describes some cases of scarlet fever in Melbourne in 1855. In one family, several typical cases occurred, only one being severe. One case was ushered in with diarrhœa and tenesmus. There had been sore throats at the school one of the boys was at, but he was not the first to be attacked.

In 1857, a discussion on the subject of scarlet fever occurred before the Med. Soc. of Vict. (*Australian Medical Journal*, Vol. ii, 1857, p. 133.) Dr. MacKenna said the epidemic had been preceded by measles; Dr. Livingstone had seen both diseases co-existent on the voyage to Australia; Dr. Tracey had seen, two years before, a severe epidemic of it in Melbourne, following measles; Dr. Fisher had not seen it so prevalent in Australia as in 1854; and W. Gilbee said that both measles and scarlet fever in Australia did not seem to require the same amount of attention as in England, children being often seen running about during its course.

Dr. O'Doherty (*Australasian Medical Gazette*, Dec., 1890, p. 53) mentions having met with malignant scarlet fever at Ipswich, Queensland, in 1865.

D. J. Thomas (*Australian Medical Journal*, Vol. xii, 1867, p. 83; discussion, p. 103), in a very interesting paper entitled "On the Recent Epidemic of Measles, Scarlet Fever, and Rubeola in Melbourne," gives some important instances of the occurrence of two of these exanthemata, within a short time of each other, in the same person. In this outbreak of the three diseases, which were still prevalent when he read his paper, he had seen rubeola followed by measles or scarlet fever and had concluded the former was a separate entity, and not a mixture or hybrid. He had also seen measles or scarlet fever following closely the one on the other. He further refers to cases without any eruption.

In speaking of the winter of 1851, he says that scarlet fever and measles were prevalent in Melbourne. His own family contracted scarlet fever; in one case, the child developed anasarca as a sequela, and during its presence the patient became suddenly collapsed, and this was followed, during the night, by the appearance of the rash of measles. In combating the collapse, croton oil was rubbed into the skin. When the measles rash had vanished, six days later, pustules from the use of this oil came out. These facts Dr. Thomas considered as tolerably good evidence "that one disease, though not annihilated, may be arrested on the advent and during the development of another." The epidemic of scarlet fever affected all ages. It was very fatal in some districts and not so in others. The extreme virulence in North Melbourne he attributed to a manure depôt; the prevalence at Emerald Hill to its being low and swampy; and at Collingwood, to parts of this suburb having similar conditions to Emerald Hill.

Some of the cases in 1866 are of great interest. A clergyman's family was attacked. The eldest daughter died from rubeola. About a week later, on November 19th, a girl of 7 developed scarlet fever and a membrane on the soft palate and fauces considered to be true diphtheria. On the 25th, measles appeared whilst the skin was desquamating. On this date, the fauces became enormously swollen, and tracheotomy had to be performed; she died six days later. Shortly after, another child contracted measles and then scarlet fever with head symptoms and died. A fourth child had scarlet fever and then measles. Finally, the clergyman himself developed scarlet fever.

In December, Dr. Thomas's coachman became ill, showing a scarlet rash between a measly eruption, with a throat intensely red and congested. Eight days later he was covered with a scarlet rash and had a white tongue with red papillæ. A week later, diarrhœa and dysentery set in, but he recovered. In the same month, a boy of 16 showed a measly rash on his forehead and face, with a red flush in his throat. Between the spots, a red rash like that of scarlet fever was visible. His face, neck and chest were uniformly covered by this; on the abdomen and upper and lower extremities, it was in extensive patches from the size of sixpence to a crown piece. There was considerable pain and confusion in the head. Ten days later his sister developed typical measles, and he himself a typical scarlet fever rash, but with no constitutional disturbance. Dr. Thomas considered that the boy had rubeola, and then scarlet fever; the sister, measles.

In another case in a man, the rash of rubeola, lasting two days, was followed on the third day by a uniform red rash lasting four days.

The Registrar-General's returns for Melbourne show that, in a population of 139,916, 214 deaths from measles and 116 from scarlet fever occurred in the latter half of this year.

During 1871, a vessel arrived at Melbourne Heads with a case of scarlet fever on board.

Separate registration of deaths from scarlet fever began in South Australia in 1856; Victoria in 1861 (the figures from 1853 to 1860 have been elsewhere obtained); New South Wales in 1875. A comparison of the tables of scarlet fever with those for measles shows that the number of deaths in years when there has been no general epidemic are considerably greater than in the case of measles. In other words, that even in the absence of a considerable epidemic there must be a large sporadic incidence of the disease. It would seem that between 1860 and 1878, in Victoria, the number of deaths, with two exceptions, always exceeded

100, and it might almost be said that with the exceptions of the years 1870 and 1871, all the other years, from 1860 to 1878, were years of epidemic in that State. Reference to the South Australian statistics and those for the other States show that extensive epidemics occurred in the years 1863 and 1864, 1875–6 and 1877. A reference to the figures for the years succeeding these shows that mild epidemics occurred in South Australia and in New South Wales, but not in Victoria, and were in each case confined to one State. Thus in 1880, 105 deaths occurred in South Australia;, in 1884–5 there was a mild epidemic in New South Wales; in 1888 there were over 100 deaths in New South Wales, and in 1893–4 there were similar increases. Comparing these various outbreaks with the outbreaks of measles, we find that the Adelaide cases from 1863–4 were preceded by a measles outbreak in 1861–2; that the outbreaks in all the States for 1875–6–7 were preceded by measles in 1874 and 1875; and that the outbreak in 1880 in South Australia was accompanied by 9 deaths in that year from measles, and 72 in the next year, whilst there was a moderate epidemic of measles in New South Wales and Victoria. The New South Wales outbreak of 1884–5 was accompanied by measles in Victoria and South Australia, but apparently not in epidemic proportions in New South Wales. In 1888, the mild NewSouth Wales epidemic was accompanied by a moderate one of measles; and the New South Wales epidemic of 1893–4 was accompanied by an extensive epidemic of measles in New South Wales, Victoria, and South Australia.

The Epidemic of 1875 *and* 1876.—The outbreak began in Victoria in July, extending nearly all over the Colony, and showing alarming virulence in some parts. In Melbourne, with a population of 62,500, 19 deaths occurred in August; 60 in September; 179 in October; and 251 in November. The disease in Melbourne and suburbs seems to have extended on to the end of September, 1876; and 1,253 deaths had occurred since August, 1875. This Victorian epidemic is said to have first appeared in West Australia.

In a paper entitled "Notes on Scarlet Fever," W. Gillbee (*Australian Medical Journal*, 1875, p. 402) deals with this epidemic. He considered that 600 to 800 cases occurred a week for November, 1875. He saw one case with a rash at first like measles, and also others with sore throats but without febrile attacks. He met with cases of desquamative nephritis. In discussing the paper, Dr. Martin thought that kidney complications were comparatively more numerous in this epidemic; he had also seen cases suddenly fatal from brain complications. Dr. Moloney, amongst the poorer class of people, found 1 death in 5 or 6; and several others spoke of its malignancy.

The vital statistics of Melbourne and suburbs from this period always contain frequent references to one or several deaths from scarlet fever for various months, but the disease does not seem to have again reached epidemic proportions in this city, though there is a reference to the occurrence of it in the suburbs of Melbourne in September, 1883.

In 1882, though the mortality tables do not indicate any excessive number of deaths for the years 1882 to 1883, the disease nevertheless appears to have been considerably prevalent. The following references occur in 1882:—Albury, Wagga Wagga, in New South Wales, Ross Creek, near Smythesdale, in Victoria, and Port Pirie, South Australia (January); Port Albert, Alexandria, and Mansfield, Victoria (February); Tumberumba, with a few cases at Lefroy in Tasmania, and Mount Gambier in South Australia (March); Ballarat and St. Arnaud (April); Ballarat Orphan Asylum, Euroa and St. Arnaud, Victoria, and Forbes, New South Wales (May); many towns in Victoria (July); Sydney (September).

In 1883, at Clunes and Gordon in Victoria (February); and Ararat (May). The immigrant ship "Roslyn Castle" arrived in Sydney with scarlet fever (March).

The Epidemic of 1884–5.—In the previous year, 1883, there is a statement that scarlet fever existed in 35 localities in Victoria (June), but the mortality as evidenced by the vital statistics cannot have been great. In 1884 the disease appeared at Wagga Wagga (March); Adelong (April); Ballarat (May); and Barmedman, New South Wales (October). In 1885 it occurred at Penrith (February); and Gulgong (May).

In 1887 cases occurred in South Australia, but apparently of mild type, since only 7 deaths occurred. The *Australasian Medical Gazette* says that, though the disease had occurred in different parts of the Colony, it had been prevented from assuming the form of an epidemic. Beechport and Millicent are noted as having been affected (October).

In 1888 the disease appeared at Ballarat (May); and Rupanyup, Victoria (November).

In 1889 a mild epidemic occurred in New South Wales, with 111 deaths, of which 24 occurred in Sydney and suburbs. Dr. A. Jefferis Turner (*Australasian Medical Gazette*, Dec., 1890, p. 65) describes the epidemic, which began at the end of this year in Brisbane and extended into 1890, with 14 deaths for the first seven months of that year. Dr. Turner's first case at the Children's Hospital was in November. The type was fairly severe. In his notes on the cases, he mentions injection of the conjunctiva, well-marked in some cases during the rash; vomiting, common at the outset, accompanied in two sisters by purging; an infant with a malignant type died in a few hours; in 3 cases there was suppuration of the sub-maxillary glands; in 1 gangrenous fauces; in 3 purulent otitis media; in 1 suppuration of the ankle-joint; and in 1 case there was nephritis and anasarca, from which the patient recovered. One patient had one-sided convulsions and paralysis (from which recovery took place) six days before the rash. This epidemic continued in Brisbane throughout the year 1890. In the same year, 41 deaths occurred in Melbourne and suburbs, and there is mention of 18 cases at Brighton (February), and numerous cases at Avoca, Victoria (October).

In 1893–4 the disease was very prevalent in Sydney, with 181 and 111 deaths, respectively. In the first half of 1893, it was estimated that there were 2,000 cases in Sydney. In the *Intercolonial Quarterly Journal of Medicine and Surgery* (Vol. i, No. 1, May, 1894, p. 384), Dr. E. A. Mackay gives notes on a "Case of Concurrent Scarlet and Enteric Fever." The patient was a woman of 22, and, besides showing a scarletiniform rash, nephritis and peeling, exhibited also unequivocal signs of typhoid fever, such as severe hæmorrhage from the bowels, rose-spots, &c. He notes that, when the hæmorrhage occurred, the albumin disappeared, and suggests in consequence that venesection might have been serviceable in relieving the kidneys if performed early in the case.

In 1896 it was prevalent in the Victorian shires of Borong, Buninjong, Euroa, Stawell, Warregal, and Yea (August). In 1897, at Zeehan, in Tasmania (January); shire of Glenelg, in Victoria (August); and Temora, in New South Wales, and Alverstone, in Tasmania (December). In 1898, it was prevalent in Tasmania, especially at Burnie (January), and Russell (May). In this year it was also prevalent in Sydney; and in June, signs of an epidemic appeared at Adelaide. In 1899 it was prevalent in Hobart (April). In 1901 it was more or less prevalent in Adelaide, a few cases occurring as far north in South Australia as Quorn. The s.s. "Gothic" arrived in Tasmania with the disease (November). In 1902, an outbreak occurred at Millicent, South Australia (April), and at Hay (May). In 1903 it was rife in New South Wales, especially the metropolis (5,358 cases reported), and during the first half of the year, 847 cases and 5 deaths were reported in South Australia. A serious outbreak occurred at Mount Morgan (December). There were 4,056 cases reported in Sydney for 1904, and 1,773 for 1905. It was very prevalent at Broken Hill in April of 1905, and in Sydney in September. It was epidemic in Adelaide in the first half of 1906, and in the latter half in Sydney, abating towards the end of the year. In December, 1908, and the beginning of 1909, it was more prevalent in Victoria than usual.

Whooping-Cough.

According to Wentworth,[1] up to 1824 not a single instance of whooping-cough in children had occurred in New South Wales.

According to Hirst,[2] whose reference is Scott (*Trans. Prov. Med. Assoc.* 1835, III, App. XII), it was imported in the third decade of the eighteenth century into Hobart (Tasmania), and became somewhat prevalent there. Thence it came to Sydney and to the western parts of the Australian continent (Milligan: *Trans. of the Calcutta Med. Soc.*, 1839, VIII, App. XI). He states it became epidemic in Tasmania and Australia several times, especially in the years 1842–3, and 1855. (Power: *Dubl. Jour. of Med. Soc.*, 1843 (March). Hall: *Trans. Epid. Soc.*, 1865, II, 74). Hall (*Aus. Med. Jour.*, 1858, p. 96) refers to the presence of whooping-cough in Tasmania in 1842 or 1843, when he caught it himself and communicated it to his children. He also records 1 death from it in the Queen's Orphan School, Hobart, in 1849, and 7 in 1855. There were 31 deaths from the disease in the district of Hobarton in 1855, all being children under 5 years of age.

We now reach the period comprised within the mortality tables attached to this paper. In this connection it should be noted that whooping-cough is not so much a disease fatal in itself, as from the complications to which it gives rise, especially broncho-pneumonia. The majority of these deaths would probably be due to this cause; and therefore it may be that the figures indicate, not so much the relative prevalence of the disease, as the prevalence of fatal complications—two factors not necessarily varying concomitantly.

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[1] W. C. Wentworth's "A Statistical Account of the British Settlements in Australasia." Vol. II, 3rd Edit., 1824, pp. 82, 927.

[2] Hirst's "Handb. of Geogr. and Hist. Path.," 1886. Vol. III, p. 32.

The figures show that in nearly every State there is a more or less constant average number of deaths after whooping-cough. In New South Wales, the maxima appeared in 1878 (367), 1899 (487), and 1907 (594). These rises are not reflected in Victoria, the highest being in 1865 (365) before the New South Wales statistics began. In South Australia, we find in 1868, 152 deaths; in 1872, 135; in 1883, 128; in 1890, 129; in 1893, 121; in 1898, 112; and in 1899, 106. Only one of these, that of 1899, corresponds with the Sydney rises. Western Australia shows the greatest number of deaths in 1907, the period of the latest New South Wales rise.

A very interesting and perhaps important feature in these mortality returns lies in the constant recurrence, at intervals of a few years, usually four to six, of periods in which the death-rate falls to perhaps half or less of that of the immediately preceding years. This fall occurs almost always in all the States, though in one or other State it may occur earlier, and be reflected thus in the year before; in fact, we have in these figures a negative picture of the prevalence of the disease as compared with the positive picture in the case of measles.

At the end of 1881 the disease was prevalent at Rydal, New South Wales, and at Windermere and Trunkwood, Victoria. In 1882 it was prevalent in Brisbane (March) and Ipswich (June); at Gundagai (April) and Sydney (18 deaths for the last quarter of the year), and in Melbourne (58 deaths in Melbourne and suburbs). During the same year, there were 9 deaths from whooping-cough during the voyage of the ship "Southesk," arriving at Moreton Bay (June). In 1884, it was prevalent at Wentworth (May); and in Victoria, at Rupanyup (November) and Wandiligong (December); and at the beginning of 1885, at Penrith, New South Wales. In 1887, there were 43 deaths in Melbourne and suburbs, and the disease was prevalent at Sandhurst at the end of the year. In 1889, 179 deaths occurred in Melbourne and suburbs; and in 1890, 44.

During 1893 and 1894, the vital statistics of Melbourne show a few deaths, beginning with 1 to 12 each month, but reaching higher figures (30 in August, 1894) towards the end of this period.

In the north-west of Tasmania, an epidemic appeared in 1895, and was prevalent at Hobart at the beginning of 1896. At the end of 1898, it was very prevalent in Sydney, and 132 deaths occurred in 1899. 17 deaths occurred in Melbourne in October, 1901; and in Sydney, 16 in October, reaching 37 in December. In 1906, 165 deaths occurred in Melbourne. In Sydney, in the beginning of 1907, a severe epidemic appeared, with over 150 deaths. This year yielded, for the whole State of New South Wales, 594 deaths from whooping-cough—by far the greatest number ever recorded for this disease for any of the States. Ramsay Smith ("Whooping-cough and Measles in South Australia," Government Printer, 1909) mentions city, suburban, and country schools affected with whooping-cough in that State in 1907. The disease seems to have been widespread and very prevalent in the latter half of the year.

TABLE of Returns of Deaths for the various Australian States.

	Measles.					Scarlet Fever.					Whooping-Cough.				
Year.	N.S.W.	Victoria.	South Australia.	Western Australia.	Tasmania.	N.S.W.	Victoria.	South Australia	Western Australia.	Tasmania.	N.S.W.	Victoria.	South Australia.	Western Australia.	Tasmania.
1853*	...	123	...	...	...	...	74	...	...	...	...	...	...	...	...
1854	...	194	...	...	...	...	63	...	...	...	...	...	...	...	...
1855	...	2	...	...	...	...	15	...	...	...	...	...	...	...	...
1856	...	6	...	...	...	...	7	3	...	...	...	...	32	...	...
1857	...	8	...	...	...	...	25	1	...	...	...	...	27	...	...
1858	...	4	2	...	...	...	29	15	...	...	...	...	28	...	...
1859	...	2	1	...	...	...	86	21	...	...	...	...	61	...	...
1860	...	274	1	...	...	...	427	10	...	...	...	...	61	...	...
1861	...	252	111	...	...	...	871	2	...	...	...	210	38	...	...
1862	...	20	104	...	...	...	416	20	...	...	197	197	22	...	...
1863	...	8	...	...	...	...	312	288	...	...	...	147	38	...	...
1864	...	7	...	...	...	...	278	510	...	...	...	25	63	...	...
1865	...	11	...	...	...	...	215	15	...	...	...	304	2	...	...
1866	...	427	...	...	...	...	462	2	...	...	...	365	...	...	...
1867	...	630	112	...	...	...	621	9	...	...	...	205	95	...	...
1868	...	24	17	...	...	...	460	9	...	3	...	243	152	...	78
1869	...	24	...	...	...	...	224	2	...	...	...	100	14	...	41
1870	...	3	1	...	...	...	24	9	...	...	...	50	...	...	...
1871	...	4	...	...	2	...	27	22	...	7	...	328	...	...	...
1872	...	7	...	...	1	...	135	26	...	3	...	227	135	...	38
1873	...	1	1	...	...	...	188	30	...	10	...	299	36	55	12
1874	...	256	280	...	...	...	121	64	...	21	...	151	8	13	39
1875	752	1,541	75	...	129	302	985	484	...	31	104	58	2	...	10
1876	35	5	5	...	1	1,097	2,240	244	...	97	17	13	10	...	3
1877	2	6	1	...	2	94	183	71	...	197	31	245	35	...	3
1878	1	5	2	...	...	23	136	28	...	7	367	291	87	...	3
1879	3	3	1	...	...	31	61	72	...	2	129	193	41	...	21
1880	270	252	9	1	...	50	26	105	...	1	132	179	38	...	54
1881	88	62	72	...	45	35	86	50	...	7	156	167	27	15	6
1882	10	15	11	...	...	39	89	34	...	20	109	108	43	...	...
1883	47	7	12	30	...	46	59	24	...	10	52	48	128	...	26
1884	45	233	139	95	1	229	34	29	...	12	201	209	53	1	11
1885	10	69	6	4	20	127	14	4	...	...	114	168	90	3	16
1886	6	20	3	...	1	88	14	13	...	...	244	209	93	61	27
1887	52	78	2	...	...	74	4	7	...	2	205	129	30	4	13
1888	218	30	1	...	...	111	21	3	...	5	35	21	9	...	...
1889	13	19	1	...	...	64	41	1	...	1	241	234	2	...	8
1890	7	1	1	...	...	67	68	3	...	1	254	98	129	...	41
1891	10	4	...	...	...	40	26	4	...	3	261	168	42	19	7
1892	2	1	...	...	...	69	26	2	...	3	225	179	12	13	2
1893	730	659	261	21	35	181	21	5	...	1	180	117	121	...	43
1894	143	32	28	6	14	111	67	35	...	1	225	262	60	4	5
1895	2	...	2	...	...	59	32	10	...	...	266	125	44	21	22
1896	4	3	...	...	1	48	44	4	...	2	100	77	17	9	10
1897	3	7	...	1	...	79	87	7	2	5	13	11	...	3	...
1898	510	671	54	34	45	83	42	15	...	7	241	61	112	8	4
1899	209	34	27	5	13	25	15	16	1	15	487	297	106	29	54
1900	10	112	2	1	...	9	6	5	1	9	106	52	29	31	20
1901	37	50	5	45	...	16	2	1	1	1	259	186	51	13	4
1902	107	50	235	19	1	61	15	...	2	22	261	187	36	23	23
1903	16	21	5	3	...	87	46	19	2	26	186	110	60	42	65
1904	21	...	...	1	...	50	23	10	2	4	147	46	25	17	26
1905	29	79	...	1	3	21	10	8	3	1	5	20	6	1	...
1906	17	7	1	3	4	42	4	12	2	...	10	247	16	11	1
1907	90	41	5	6	1	26	2	5	...	...	594	128	73	103	47
1908	32	20	14	27	4	40	21	...	2	...	58	68	18	20	23
1909	11	4	1	9	2	30	42	...	...	2	17	169	20	3	2
1910	99	...	...	...	...	23	...	...	...	...	174	...	...	...	...

NOTE.—I have been unable to obtain the returns for Queensland. * Six months.

In conclusion, I would like to thank those who have aided me in collating this information, more especially that part dealing with the mortality statistics.

MALARIA FEVER : ITS CAUSE, CURE, AND PREVENTION.

By A. E. Finckh, M.B., C.M. (Syd.)

(A guide to those in Authority in Malaria-stricken Districts.)

Based mainly upon the teachings of the Hamburg Institute for Tropical Diseases, Hamburg.

Malaria fever is an infectious disease of man, due to the presence in the blood of a microscopical animal parasite, belonging to the group of protozoons. It is a disease occurring only in tropical or sub-tropical countries, or after a sojourn therein. It is characterised by intermittent attacks of fever, usually of great regularity, occurring, according to the type, of which there are three, daily, every second day, or every third day.

During the attack there is headache, often vomiting, and weakness in the heart's action. The disease is accompanied by progressive anæmia and enlargement of spleen and liver, and the patient's condition is one of general weakness and apathy. The disease is not always easy of diagnosis, indeed, it is only the detection, by means of the microscope, of the parasite in the patient's blood which makes a diagnosis absolutely certain. Almost any febrile condition may simulate malarial fever, but it is a wise plan to look upon every febrile condition in the tropics or after a sojourn there as malaria, and to treat it as such, for, with our present knowledge of the therapeutics in malaria, it can be said that any intermittent fever which does not yield to quinine is not malarial, always provided that this drug is administered on scientific lines. Such yielding to quinine will take place within four or five days, so that no great harm could arise, and much time may be gained, by making such an experimental use of the drug a routine practice.

Etiology.

There is hardly a disease of which the etiology has been worked out with such exactness as that of malaria. The work done by such men as Laveran, Celli, Manson, Koch, and Nocht, is a triumph in modern pathological investigation. What they have taught us, as regards the causation and the spread of the disease, together with their teachings in respect to the drug treatment, has enabled us to successfully stamp out this once so dreaded malady.

Nor is there another disease, a thorough knowledge of the causation of which suggests to such an extent what steps should be taken regarding the cure and prevention of the disease. And whosoever may be concerned with the treatment of malarial fever, and especially with the eradication of the disease, must, above all, have a thorough knowledge of these causal factors.

Indeed, our knowledge in this respect is such that it has been said that in no district which is under official supervision, and where it is possible to issue official instructions, should malarial fever be any longer tolerated; every Government official, and every member of any institution, commercial or otherwise, can now be supplied with instructions so definite that if they are carefully carried out, malarial fever will not occur, and every official might reasonably be reprimanded if he becomes infected, because it simply means that he has neglected to carry out the instructions that have been issued to him.

It is with the intention of making known instructions of this kind that this paper has been written.

Malarial fever is due to a parasite in the patient's blood; it lives in, and ultimately destroys, the red blood corpuscle, causing thereby the anæmia which is so characteristic of the disease. This organism undergoes, like so many of these lowly-organised parasites, a very remarkable cycle in its life-history, so that it is met with in its various forms in various situations of its hosts. In the form above-mentioned, as destroying the red cells, it is known as the schizont. It grows within the red cell at the expense of the latter, and ultimately undergoes spore formation. The spores, through the breaking up of the red cell, are scattered throughout the blood; they are known as merozoites. These make their way into other red cells to develop into schizonts, and to again break up into merozoites. It is this breaking up into merozoites which is heralded by the febrile attack so characteristic of the disease, and according to the type of the organism—of which there are three—spore formation takes place at different intervals. In plasmodium vivax in 48 hours; in plasmodium malariæ in 72 hours; and in plasmodium immaculatum, also in 48 hours, giving a definite fever curve, with intermissions of one and two days, respectively, without a rise in temperature. In this manner reproduction of the parasite takes place asexually by simple spore formation; on the other hand, there is also a sexual reproduction which is conditioned by the development, out of certain of the spores (merozoites), of sexually mature, female and male, individuals, the so-called gametes. These, however, cannot give rise to a new generation unless taken into the alimentary canal of a certain kind of mosquito—the anopheles. Fertilisation takes place in the stomach of the mosquito; the resulting ookinet penetrates into the wall of the stomach, and there remains as the oocyst. Sporulation then takes place, the cyst breaking up into the so-called sporozoites, which penetrate into the body cavity, reaching the blood-stream, and finally passing with the lymph into the salivary glands. Then, when the infected mosquito bites a human being, the sporozoites are deposited into the blood-stream, and there make their way into the red cells and develop into the above-mentioned schizont with which our cycle began.

In this life-history of the plasmodium, the following points, as regards time, are of importance: the inoculation period is from ten to fourteen days in severe infections; however, it may be shorter, five to six days: that is, between the time of having been bitten by an infected mosquito and the appearance of the first attack of fever, five to fourteen days may elapse, but it must be kept in mind that the first attack or attacks may be of so mild a nature as to be totally overlooked. The mosquito, after having imbibed the blood of a malarial patient, and provided that such blood contained gametes, is not capable of transmitting the disease before the tenth day, since the development from gamete into sporozoite within the mosquito requires at least ten days. Another point of importance, and one which explains the occasional outbreak of latent malaria, is the fact that while ordinarily the gametes cannot give rise to new generations without the passage through the mosquito, yet the female gamete may, after the lapse of considerable time, regain the capacity of breaking up into merozoites like the schizont; this explains the possible occurrence of the fever after a considerable lapse of time after the possibility of infection has passed.

It may seem out of place in a practical discussion of this kind, to give such detail as the above life-cycle; however, a knowledge thereof is essential if we wish to base our treatment, and our steps towards the prevention of the disease, upon etiological facts. The above description has made us acquainted with the following facts:—Without the plasmodium there can be no malarial fever; without the mosquito there can be no spread of the disease; the presence of the anopheles in any particular district in itself does not mean that malarial fever is also present; there is no danger of infection from man to man except in the presence of the anopheles to transmit the disease. The attacks of the fever are due to the breaking up of the parasite into spores. Hence, if the parasite cannot be found in the blood during a febrile attack, the condition is not due to malaria; during the none-febrile stage of the disease the gametes are to be looked for.

A description of the particular mosquito may not be out of place, so as to enable anybody, who wishes to do so, to ascertain first if the right kind of mosquito is present in any locality before one would, for instance, initiate an elaborate scheme for its destruction. This particular anopheles is distinguished from the culex by the following features:—In a sitting position, anopheles is almost perpendicular to the surface on which it is resting; its body and head form almost a straight line. Culex, on the other hand, sits horizontally, and the body describes a curve. The wings of the anopheles have black edges, not so in culex; the palpi in anopheles are of the same length as the proboscis; in culex they are much shorter than the proboscis. The antennæ in anopheles are shorter than the tentacles; in culex they are longer. It is only the female mosquito which bites; the male is recognised by its feather-like antennæ, in the female they are brushy. The larvæ are also easily distinguished; that of anopheles rests horizontally under the surface of the water; in culex it hangs down perpendicularly. The ova of anopheles are deposited into the water singly, and they float; those of culex are deposited in masses which assume a boat-shaped form, if they are deposited singly they sink to the bottom.

Infection.

Infection by the disease only takes place through the bite of the anopheles. If the blood of one malarial-infected human being, containing merozoites or gametes, were to pass directly into the circulation of another infection, of course, would also take place. However, such a mode of transmission must be extremely rare. As regards the biting anopheles, the conditions are that such an insect has sucked the blood of a malarial patient, and that at least ten days have elapsed since such act, and that both male and female gametes have been taken into the alimentary tract of the mosquito during the act of biting.

Treatment.

When treating malarial fever we are in the happy position, not too frequently occurring in medicine, that we have at our disposal a drug which we use, not empirically, but with the definite object of removing the cause of the disease; that drug is quinine. Its destructive action upon the malarial parasite circulating in the blood is so certain that, as has been remarked when discussing the diagnosis of the disease, any febrile condition which does not yield to quinine can be safely looked upon as not being caused by the malarial parasite. It is only in extremely rare cases that the disease will not yield to this drug or that there exists in the patient a certain idiosyncrasy which will not allow of the administration of this drug, and even in such supposed refractory cases, a carefully specified administration of the drug will bring about the desired end.

In the administration of the drug several very important questions have to be discussed, for it is by no means a matter of indifference as to what amount of the drug is given or in what form it is administered.

In the past many valuable lives have been lost because such rules and regulations have not been observed. The first of the rules is, that a most careful selection of the form in which the quinine is given. When we consider that sulphate of quinine is soluble in 800 times its weight in water only, whereas the bi-hydro-chloride is soluble in its own weight, the importance of never giving the sulphate in powder or in tabloids should immediately suggest itself. Of the various quinine salts the following shows their respective solubility:—

Sulphate of quinine	1 in 800
Bi-sulphate	1 in 10
Hydro-chloride	1 in 35
Bi-hydro-chloride	1 in 1

It should, therefore, be made a rule never to give any sort of quinine in solid form except the bi-hydro-chloride. If the others are employed, they must be given in solution.

The next consideration is that of the administration of the drug in the form of pills, tabloids, &c. The administration of quinine in these forms should be altogether avoided, because there is always the danger of these pills, &c., passing through the body undissolved. If one is obliged to use them, then they should be tested with regard to their solubility by placing in water—if within a few minutes they do not show signs of breaking up, they must be looked upon as unfit for administration. The method of taking the drug wrapped up in cigarette paper must be absolutely condemned. It is true numbers of people have great faith in this manner of taking it, and they say that they are able to take quite large doses of it without experiencing any bad after-effects. But they forget entirely that the cigarette paper will pass undissolved and without unfolding through the body and that not a grain of the drug has had an opportunity of being absorbed.

The Dosage.

The method of administration to be detailed here is that employed at the Hamburg Institute for Tropical Diseases. Professor Nocht states that since the introduction of this somewhat complicated method, their results have been such, both as regards the cure and avoidance of undesirable after-effects, that the same cannot be too highly recommended. The amount per day for an adult is 15 grains; in children, 1·5 grains per day for each year. This daily amount is administered in five separate doses; thus, for an adult, 3 grains five times daily. The treatment is begun as soon as the disease is diagnosed or suspected, regardless of the time of the febrile attack.

Dosage.

15 grains daily for 6 days,
    Omit quinine treatment for 2 days.
15 grains daily for 3 days,
    Omit quinine treatment for 3 days.
15 grains daily for 3 days,
    Omit quinine treatment for 4 days.
15 grains daily for 3 days,
    Omit quinine treatment for 5 days.
15 grains daily for 3 days,
    Omit quinine treatment for 6 days.
15 grains daily for 3 days,
    Omit quinine treatment for 7 days.
15 grains daily for 3 days,
    Omit quinine treatment for 7 days.
15 grains daily for 3 days,
    Omit quinine treatment for 7 days.
15 grains daily for 3 days,
    Omit quinine treatment for 7 days.

If at the end of this time the patient is no longer exposed to infection he is regarded as cured. Should any relapse take place, or a new infection occur, the whole scheme must be repeated. Anybody, after such a cure, still remaining in a malarial-infected district, must be treated prophylactically, as to be described in the next chapter.

Prophylaxis.

It is possible to prevent infection with malarial fever, provided there is sufficient determination, for it is necessary to administer a certain daily dose of quinine during the whole of the stay within an infected area. Such prophylactic dose for an adult is 5 grains daily, which is taken in one dose, or better still, in two half doses; for children the dose is ½ grain for each year. Thus, the child of three years will get 1½ grains. After leaving the infected area, it is necessary to continue taking this prophylactic dose for a further period of two months.

When dealing with schools, institutions, plantations, &c., it is always advisable to have the administration of the drug properly supervised, otherwise it will be found that the results will not be satisfactory, since even people who ought to know better will neglect in a most careless fashion the routine of taking the drug. The most satisfactory method is to make up a considerable stock of the drug in solution, and cause the patients to come daily to the dispensary or office where the drug is swallowed in the presence of the official.

Idiosyncrasy.

Occasionally one meets people who state that they cannot take quinine on account of disagreeable head symptoms, such as deafness, ringing in the ears, and fulness in the head; however, if the above method of administration be adopted, and if, where such symptoms do occur, the daily dose is reduced and then gradually increased, most people will learn to tolerate the drug. In some cases it is necessary to begin with the adult daily dose as small as 3 grains.

It must be stated here that, especially in malarial districts, quinine is often credited with various disadvantages which it certainly does not possess. In malaria, before one would feel justified in withholding the drug, a most pronounced idiosyncrasy must be present. No doubt the senseless abuse of the drug in the tropics, where some people cheerfully boast of the heroic doses they are prepared to take, must do much harm. But this must not be weighed in the scale against the use of the drug.

Black-water Fever.

For all practical purposes we are justified in looking upon black-water fever merely as a complex of symptoms, the chief of which is the appearance of blood in the urine. There are absolutely no proofs that the condition is a disease in itself with its own protozoon causator. The experience of most careful investigators has taught us that the condition is a specific intolerance against quinine or some anti-pyretic, occurring almost exclusively in patients suffering from malaria, and with over 90 per cent. of such patients the malarial infection has been acute and extending over several months.

The treatment of black-water fever is that of malaria. But before the quinine treatment can be satisfactorily proceeded with, it is necessary to determine experimentally what dose of quinine the patient will tolerate. It is necessary to begin with a small adult dose, say, of 3 grains; if such does not produce a rise in temperature, icterus, albuminuria, or hæmoglobinuria, the dose is increased after two or three days to 4 grains, and again the result awaited. By a gradual working up it will be found that within two or three months the adult dose of 15 grains will be tolerated.

Should it be known that a patient, about to be treated for malaria, is liable to suffer from black-water fever, the initial dose of quinine will be small, and will be gradually increased in order to avoid the hæmoglobinuria which a sudden large dose of quinine would cause.

General Treatment.

During the attack, and immediately afterwards, the patient is treated on general principles, and with such tonics, including arsenic and iron, which promote a new-formation of blood.

Prevention.

The prevention of the disease can be discussed under the following headings:—

1. The protection of the population from the bites of the mosquito.
2. The destruction of the mosquito.

Whoever is able to protect himself successfully from the bites of the mosquito will never become infected with malarial fever. This may not seem an easy task. It may at first appear hopeless, and many people will laugh at the idea of even making such an attempt, but experience, and the history of such places as Ishmailia, on the Suez Canal, and of the Panama Canal, are sufficient proof that the disease can actually be stamped out.

When we consider that it is only the female mosquito which bites, and that it has been shown that perhaps not more than 2 per cent. of them are capable of transmitting the disease, it will be seen that even the destruction of a small number regularly carried out, may do a good deal in checking the disease. When making a plan of campaign against the mosquito, it is useful to bear in mind its habits. The mosquito cannot breed without water, the eggs must be deposited into it, and the larvæ live in it. Hence, the first step would be to remove all possible breeding-places. The water preferred by the mosquito is usually stagnant, small lakes and pools, or even an empty jam-tin, carelessly thrown out and filled by the next shower of rain, may become the birth-place of thousands of mosquitoes. Therefore, we must do away with all such likely breeding-places by removing them, by filling them in, or by surface drainage. Larger areas of water can be effectively treated with kerosene, about 1 pint being required for each 8 square yards of surface, and repeated every month or so. This, by forming a thin layer on the surface of the water, prevents the mosquito from depositing its eggs in the water, and also prevents the larvæ from rising to the surface in order to breathe. All tanks and other receptacles for drinking-water must be carefully protected by means of netting or other covering to prevent the mosquito from reaching it.

Since the mosquito does not travel any great distance, it will be found that if the above precautions are observed and a few hundred yards surrounding a building are thus kept clean, a very perceptible difference as regards the annoyance caused by the mosquitoes will be noticed.

The anopheles rarely bite through the day, thus differing from other species of mosquitoes. In daytime they hide away in any dark corner or sheltered place, or under low bushes; therefore, all such likely resting-places must be removed, and low shrubs, &c., in the immediate neighbourhood of the dwelling should be cut down. Large trees, provided the foliage does not reach to near the ground, will not be found to harbour mosquitoes. The anopheles avoid light, airy and well-ventilated places, hence everything should be done to leave all vacant spaces, such as under houses, free to access of air and light. The mosquito will not travel against wind, therefore dwellings should be built so as to lie away from water in the direction against the prevailing winds. All dark places, such as under the houses, should be regularly fumigated early during the afternoon while the mosquitoes are resting. Making use of the fact that mosquitoes prefer dark places to rest in during the day, very effective traps can be made in the shape of dark holes dug somewhat slantingly into the ground. These, especially when made damp by pouring in some water, become very favourite resting-places for the mosquitoes, and large numbers can be killed daily by smoking out as suggested above.

Personal Protection.

As regards personal protection, much can be done by wearing suitable netting, and by protecting hands and ankles during the evening hours. A mosquito-proof room, or verandah is in itself such a comfort that it is well worth the trouble and expense of making one. The question of stuffiness somewhat complained of in the tropics is easily overcome by proper ventilation.

Another important point, usually neglected, is the protection of all malarial patients from the mosquitoes. All natives, who are likely to be harbouring the malarial parasite, should not be allowed to spend the evenings or nights near the dwellings of Europeans, and every patient should, during the evening and night, be carefully protected by netting so as to prevent him from infecting mosquitoes which in their turn would spread the disease; this is especially important in districts where the disease is not very prevalent but where the mosquitoes are in abundance.

Much watchfulness and ingenuity is required if the above suggestions regarding the cure and prevention of the disease are to be fully effective. It requires for each locality a well-thought-out plan of operation which must be persistently carried out.

This article has been written with the distinct object of making known only so much of the cause and spread of the disease as will be required to employ intelligently such modern methods which are proving everywhere of such success. Thousands of lives are being annually preserved, and many localities, which previously have been looked upon as almost uninhabitable for Europeans, are now being used as health-resorts, by the adoption of the methods described in these pages. It is hoped that it may serve as a guide to those who are interested in the matter of rendering life more endurable in the tropics.

INFECTION OF THE URINARY TRACT BY COLON BACILLI.

(A series of 34 cases treated at the R.P.A. Hospital, Sydney.)

By Cecil Purser, B.A., M.B., C.M., Senior Honorary Physician, and Neville J. Davis, M.B., Resident Physician, R.P.A. Hospital, Sydney.

Up till the last few years, urine which was acid in reaction and showed the presence of both albumen and pus was considered by the general practitioner to be the secretion of a kidney, which was generally the seat of tubercular disease. Since then, however, bacteriological methods have been more widely applied to clinical medicine, and no urine analysis is considered complete at the present time without such an examination. Under the old régime when the acid urine was examined for the bacillus tuberculosis it was found absent in many specimens, even after several examinations. Then arose the doubt that cases with acid urine and pus and albumen present, did not always owe their origin to tubercular kidney disease, and it was shown that the colon bacillus could invade the urinary tract and cause an acute inflammation of it. A recognition of this fact cleared up a large number of cases which previously had been unsatisfactory, from the point of view of diagnosis equally as from the point of treatment. It took us some time to realise this fact, but when we did it was found that the majority of cases in which acid pyuria occurred were of *Bacillus coli* origin. It was noticed that tubercular disease of the urinary tract was not of very common occurrence, and was greatly outnumbered by the inflammations of the urinary tract due to the colon bacillus.

During the past eighteen months we have collected thirty-four cases of inflammation of the urinary tract due undoubtedly to the colon bacillus. However, the severity of these attacks is not always the same, and of our thirty-four cases eight have been mild or sub-acute inflammations. The manner in which we have arbitrarily classified these is as follows:—In the mild or sub-acute cases a patient did not have much rise of temperature, never going above 100° F.; the pain was in every case described as dull and aching rather than sharp and stabbing; but, on the other hand, these sub-acute cases have been more resistant to treatment and more protracted in their course.

Ætiology.—With regard to the ætiology of the disease two factors—exciting and predisposing—have to be considered. In our twenty-six acute cases nineteen of the patients gave a history of feeling cold or getting wet and subsequently chilled. This indefinite " chill " occurred always within twenty-four hours of the onset of the pain. Consequently we may assume that in the acute cases the chill lowers the resisting power of the body, and the colon bacillus which is already present within the body gains the upper hand. In the other seven acute cases no definite cause could be assigned by the patient. There are many predisposing factors in the onset of the disease, viz.:—

Seasonal.—In our series there was no marked preponderance of cases in any one month or season. In the winter of 1910 there were only nine cases, seven of which were acute.

Age.—The oldest person in our series was 53 years, and the youngest 19 years, whilst the average age was 28½ years. There were fifteen cases in which the age of the patient varied from 19 to 28 years, whilst there were eleven cases in which the ages varied from 29 to 38 years. So that it may be concluded from our series that it is much more common to find an inflammation of the urinary tract due to the colon bacillus in patients under 40 years than over that age. Another point we have noticed is that when it does occur after 40 years of age it is frequently of the sub-acute variety.

Sex.—This was a most striking point in our series of cases, and the conclusion we arrived at was that the disease was common with women as compared with men. Of our thirty-four cases twenty-eight, or 82 per cent., were women. On a further analysis of the series we found that of these twenty-eight women only four, or 14 per cent., were unmarried. This led us to the belief that the disease occurred primarily amongst married women. We looked carefully for the reason of this, and we are of the opinion that it is due to a combination of causes which are mentioned later.

Constipation.—In a large number of our cases we took care to inquire into the state of the patients' bowels previous to the onset of the illness, and our investigations led us to a conclusion for which we were not unprepared. In thirteen of the cases our notes did not include the condition of the patients' bowels before they became ill, but of the remaining twenty-one, sixteen patients said that they were definitely constipated just before the onset of the illness. This constipation they said was more obstinate than usual with them. In one case the patients only had two movements of the bowels in eleven days. Following upon these inquiries we are able to say that 76 per cent. of these cases had varying degrees of coprostasis. Under these conditions the colon bacillus, already an inhabitant of the intestine, was given an increased field for activity. For this reason we are inclined to think that there was definite relationship between coprostasis and infection of the urinary tract by the colon bacillus. Of the remaining five cases two suffered with diarrhœa, one rather severe, there being eight stools daily. So that, in these cases, the action of the colon bacillus was intensified.. It would therefore seem rather more than a coincidence that 85 per cent. of the cases in which the state of the bowels previous to the onset of the illness could be ascertained had some alteration from the normal average daily movement.

Another point which drew our attention, was that of the twenty-four cases who were married women six, or 25 per cent., were pregnant. In three of these six cases the fœtus was 2 months old, and in the remaining three the fœtus was respectively 5, 6, and 7 months old. So that it would seem that pregnancy adds to the risk of the colon bacillus invading the urinary tract.

Two of our cases occurred after the patient had been curetted, and a catheter had subsequently to be passed. In each case the onset of the illness occurred within forty-eight hours of catheterisation. Of course this may have been only a coincidence, since in each case the patient said she had been constipated during the preceding week.

Summing up, we are inclined to think the lowering of the patients' resisting power by that indefinite "chill" is the existing cause, whilst constipation and pregnancy prepare the way if an exciting cause is present. Then, again, it is commoner in the middle period of life, and more common in women than men, and much more so amongst the married than the single.

Symptoms.—The patient may not be able to describe any definite symptoms, but just complains of a general lassitude. One of the most constant symptoms is *pain.* It occurred in every case of our series, but an analysis of its intensity of location afforded us interesting material for thought. In only one case did it occur in the upper part of the abdomen, and that was in the right side, under the costal margin. In the remaining thirty-three cases pain was always present, and in nearly every case the first symptom noticed in the disease. In ten cases the patient stated that it was equally severe in both lumbar regions. Fifteen patients located the maximum point of intensity in the right lumbar region, and in eight cases the left lumbar region was the seat of the pain. From these observations it would seem that the right urinary tract was affected sixteen times, the left tract eight times, and both in ten cases. Consequently, we have inferred that the right urinary tract is more frequently the seat of inflammation than the left.

Tenderness.—There is quite definite tenderness on palpation over the kidneys, the course of the ureters, and the bladder, and it may be so deceiving as to simulate appendicitis when the tenderness is situated in the right side.

Frequency of micturition.—In nearly every case the patient said that they passed the urine much more frequently than usual, but as one put it, it was little more than desire as they passed so little. They also said that the act of micturition involved more or less pain. The pain was generally of a smarting character, and seemed to be in the urethra.

Vomiting is often present at the beginning of the illness. It is never very protracted. In our series only twelve, or 35 per cent., gave a history of vomiting. Of these cases very few had more than three attacks. The vomiting did not have any special relationship to the taking of food, and in many cases the vomit consisted of greenish-yellow fluid, whilst in others it was food that had been previously just ingested. But there was not a case in our series in which the patient was unable to retain food that she had swallowed. Vomiting did not occur in any of our eight sub-acute cases.

Temperature.—Let us consider the course of the temperature first in the acute cases. There was frequently a rigor at the onset, and the temperature suddenly rose, and after reaching its maximum gradually fell by lysis and became sub-normal before settling down. The time before the temperature settled down varied considerably. The shortest time was six days and the longest thirty days, whilst the average number of days was between fifteen and sixteen. After coming under treatment, however, the time before the temperature became normal had varied between two and twelve days, with an average of seven days. We shall speak about the effects the various kind of treatment had upon the temperature later. At the onset of the illness the temperature is high. Amongst our twenty-six acute cases the highest point which the thermometer registered was 104·6° F., whilst in three other cases the maximum was 104·4° F. Altogether there were eight cases in which the temperature rose to 104° F., or higher. Taking the highest point the thermometer registered in each individual acute case we found that the average was 102·8° F. In the other eight cases, which were sub-acute, the temperature never arose above 100° F.

Pulse.—The pulse rate did not rise *pari passu* with the temperature, and in very few cases were there 120 beats of a minute in any stage of the disease.

Analysis of the urine.—In every case of our series the urine gave an *acid* reaction. On testing for albumen, in all but one case, we noticed the presence of a cloud of albumen commensurate with the pus present. In that case where it was absent pus cells were only found microscopically, and no pus could be detected with the naked eye. The urine was always more or less opalescent, without much deposit. The specific gravity varied from 1,008 to 1,028, and did not present much information. On plating out a sterile catheter specimen of the urine on agar we grew in thirty-one of our cases a bacillus of the colon group, whilst in the other three cases we did not get an organism at the first examination, but it grew on culturing a second time. We examined the urine at intervals of three to four days after the acute stage had subsided and we found that with the fall of the temperature to normal the pus diminished in amount and disappeared from the urine altogether within a week or so. In some cases we tried to culture the organism when the pus cells could not be detected microscopically, but we found our agar plates sterile.

We have seen cases, however, not included in our series in which a pure culture of the colon bacillus was obtained some weeks after patients were apparently well, and pus could not be detected. We did not adopt the procedure of repeatedly culturing the urine in all our cases, but only in some of the later ones of our series. In our sub-acute cases the pus was much longer before it finally disappeared though present only in small quantities. We frequently found the pus would not be present in the urine one day during the acute stages of the disease, and yet be found next day. But it was never absent for three days at a time, and then returned the following day. We were told that although the temperature fell to normal and the pus cleared up, still relapses occurred and the temperature rose again and pus reappeared in the urine. We paid attention to this point and our experience was that in twenty-eight of our cases there was no relapse. In six of the cases, however, four of which were the sub-acute variety, we did get further trouble after the urine had cleared up and the temperature become normal. In two cases there was one rise of temperature after it had settled down. In one case the temperature went to 100° F. six days after the patient had settled down, and only remained up for thirty-six hours, and the pus was detected in the urine microscopically for three or four days. In the second case, however, we got a true relapse or reinfection, because five days after the temperature came down to normal and the day after the pus had disappeared from the urine, the temperature went up and did not subside again for eighteen days. The highest point reached during this attack was 103·8° F. The urine cleared up five days later, and the patient was kept under observation for three weeks without further relapse occurring. In a third case three days after the temperature had become normal, but while there was still pus present in the urine in a small amount, the temperature rose to 104·8° F., and gradually settled down. The attack lasted five days. Four days later the temperature again went up to 100·6° F., but was normal in thirty-six hours, and the urine cleared up again the following week. The patient became well without further relapse. We had another case exactly similar to this. In these two cases the urine had not cleared before the temperature rose the second time, so that it was more a recrudescence than a relapse. In another case we had three separate rises of temperature after the first attack had cleared up. The first occurred eleven days after the chart became normal, when the urine was clear. Not any of these exacerbations were acute or prolonged, and the patient cleared up at the end of seven weeks. In one of our sub-acute cases we had six different rises of temperature at varying periods after the initial attack had cleared up. This was a patient who had been treated on autogenous vaccine from the first.

As far as we could we kept our patients under our observation for a considerable time after the urine had cleared, but in seven of our cases we could not definitely say they were cured, because they went away within a week of the time that their urine cleared. But exclusive of these our remaining cases were kept under observation and the urine examined for varying periods, up to seven weeks, and we considered all cured. The average length of time they remained under observation was three weeks, and we did not get any fresh appearance of pus in the urine.

Diagnosis.—There were three cases in which the diagnosis was only arrived at after operative measures had eliminated other diseases. In one case a patient had pain in the right iliac region, with vomiting, constipation, tenderness, and a rise of temperature, with a history of having been sick for two days. The surgeon operated with a provisional diagnosis of appendicitis, but the appendix was found quite healthy, whilst the pelvis of the right kidney was swollen, and next day a copious pyuria was detected. In the remaining two cases there was a doubt about a renal calculus. Although the X-rays failed to discover anything, the surgeon performed a nephrotomy, but failed to find any stone, and the pelvis of the kidney was enlarged and slightly œdematous.

The combination of pain in either side or both sides with previous constipation, tenderness, and frequency of micturition, does not offer a very striking picture, but the last symptom should put one on the right path, and an examination of the urine, which resulted in an acid reaction with pus and albumen present, should at once make a diagnosis of colon bacilli infection of the urinary tract probable. Then a culture of the urine would settle the matter.

Treatment.—The pathological indications for treatment are clear, and we have found that the cases we tried following out these indications gave us best results. The urine is acid, and it is practically proved that colon bacillus will grow in an acid medium, but perishes in an alkaline one. Therefore, while the urine is acid the organism can still exist, whilst any drugs that make the urine alkaline bring the disease to a conclusion. Amongst our cases we tried various forms of drug treatment with a view to arriving at the best one. Although the number of cases tried were too small for us to make any definite statement, still our results show which are the best. The drugs we used were citrate of potash in 20 to 30 grains, doses every three or four hours; urotropine, 5 to 10 grains, every four hours; and autogenous vaccine. We used all the combinations of these three forms of treatment, either separately, or two or three at a time; using one till the temperature dropped, and a combination afterwards. To explain more clearly we used citrate of potash until the temperature became normal, and then added urotropine. We gave urotropine till the temperature fell, and then added citrate of potash. We gave both citrate of potash and urotropine all through the illness. In some we used citrate of potash alone, whilst in others the patient was ordered urotropine all the time and also autogenous vaccine by itself. Then we gave other patients urotropine and autogenous vaccine combined; and still, in others, we gave all three together. Viewing these results we find that we gave citrate of potash alone in four cases, and the temperature fell to normal in an average of eleven days, and no relapses occurred. We got the next best results from a mixture containing citrate of potash and urotropine from the beginning, and in these three cases the temperature became normal in thirteen days, and no relapse occurred. In five cases urotropine alone was given, and although the temperature fell to normal in fourteen days, we had two relapses, one case relapsing three times. In three cases we gave citrate of potash until the temperature came down and then added urotropine. The temperature fell in fourteen days, but one case relapsed twice. In five cases where we gave urotropine till the temperature came down and added citrate of potash after that, the temperature took nineteen days to subside, but there were no relapses. When in three cases we put the patient on urotropine, and gave autogenous vaccine from the start, we found the temperature came down in eighteen days, but in two relapses occurred, one of which had six definite relapses. When we gave vaccine alone, in three cases the temperature did not drop to normal for a month, and one case relapsed twice. When we gave all three in combination we did not have any relapses, but it took twenty days for the temperature to reach normal.

From these observations we are of the opinion that citrate of potash is the best treatment, and we noticed that the urine became alkaline one or two days previous to the temperature becoming normal. Urotropine, although a good urinary antiseptic, keeps the urine acid, and there may be not sufficient formalin liberated to preserve antisepsis in the urinary tract, and then the organism has a favourable medium provided for its growth. This might account for relapses. We have had rather disappointing results from our vaccine therapy, as the disease was inclined to become chronic. We gave doses of 30,000,000 bacilli every five days, and the largest dose we reached was 180,000,000. Since it has been reported by some authorities that large doses of vaccine—100,000,000 and upwards—give the best results. As regards vaccine therapy, it is quite possible that had we adopted this same large dosage our results may have been more favourable.

INTRAVENOUS INJECTIONS.

By Philipp Heymann, M.D., Gayndah, Queensland.

It is a matter of quite common occurrence that the expressions "Infusion" and "Injection" are confused the one with the other in such a way that the term "injection" is ordinarily used instead of "infusion." In two articles published quite recently, viz., in the March edition of the *Australasian Medical Gazette*, dealing with the administration of salvarsan, the respective writers speak of injections where, from their description, it is evident that they have performed and really mean infusions. "Infusion" means the introduction of liquids into the subcutaneous connective tissue or any other tissue or into cavities of the body under the pressure of their own gravity only, which may be modified by varying the height of the vessel containing the liquid. Whereas in an injection the entrance of the liquid takes place under more or less high pressure, according to the force used to drive the liquid into the body and to the resistance offered by the tissues, but under, at any rate, higher pressure than the liquid's gravity. For an infusion one uses large quantities of low concentration; for an injection, small quantities of high concentration. Nowadays subcutaneous injections and intravenous infusions are the rule; subcutaneous infusions are rarely practised, and still more rarely intravenous injections.

Intravenous infusions were performed as early as the seventeenth century. The first publications on the subject date from the years 1665 and 1668. These infusions were effected in this fashion—the chosen vein was exposed by incision, opened by means of scissors, and then ligatured, after the liquid had entered. Later on, the method was abandoned, firstly, because in performing it in a careless way, and owing to the lack of knowledge of antisepsis or asepsis, disagreeable consequences were experienced, and secondly, because with the introduction of the subcutaneous injection, the intravenous administration appeared superfluous.

The intravenous injection, however, is of recent date. The first injection of this kind on a human subject was performed on the 27th May, 1881, by Professor Landerer. Baccelli, who frequently is credited with its discovery or initiation, obtained from Landerer a reprint of his article and made the injection according to his directions. Now, although meanwhile—it is true within certain well-defined limits—intravenous injections have been practised, and although it has been demonstrated thus that the method is devoid of any general or special danger, there is still at present a certain aversion among medical men from availing themselves of this method.

The principal objections are the possibility of septic infection, and of air invading the circulation. Both dangers can be obviated with certainty. Moreover, it is said that a small surplus of the injected drug may have dangerous results, but this objection may be raised against any other mode of administration, and does not destroy the truth of the possibility of eliminating all risk. Finally, it is pointed out that the composition of the blood may be interfered with, that the wall of the vein may be permanently damaged, &c. Objections of this kind indeed require grave consideration, and we have to take care that the drugs or their solutions to be used for intravenous injection do not cause coagulation of the blood or obstruction in the capillaries, that they do not damage in any way whatever the corpuscular elements of the blood, and that they do not affect the physiological function of the inner layer of the venous wall—quite apart altogether from the pharmaco dynamic properties of the drug. A solution that fulfils these requirements may be used for intravenous injection with safety. Sometimes one finds pointed out the technical difficulties that are met with in using this method. There are really none. The injection can be made quite easily in the consulting room. In several cases I have performed it in the private residence of the patient. Of course, like every new manipulation to which one is unaccustomed, it requires practice.

It is essential to render the vein chosen for the injection—usually one of the superficial veins at the elbow—prominent, which is easily done by applying an elastic bandage around the upper arm. The arm, resting on a cushion, is, at the elbow, strongly extended, rather, if anything, over-extended. The syringe, filled with the sterilised solution, from which the air-bubbles have been expelled, is held in such a manner that the point of the needle is directed towards the patient's shoulder, while the long axis of the syringe lies parallel to that of the vein. The needle is then, at a very acute angle (almost, in fact, parallel-wise), thrust into the lumen of the vein, and the solution injected slowly and evenly. One can make gentle suction before injecting, and when blood flows into the syringe, the operator is certain he is in the lumen of the vein. But usually one feels that the needle is in a cavity, and under favourable conditions one can move the needle within the cavity up and down. If the suction fails to draw blood into the syringe, or if after the first drops have been injected, a swelling or prominence occurs near the vein, in the adjacent tissue, the syringe should be removed and re-introduced. For this event it is advisable to put more of the solution into the syringe than one has the intention of actually injecting. Or one can inject in the first instance physiological saline solution, and if no prominence be produced, follow this up by injecting the effective solution; finally one may re-inject some saline solution. By means of a special contrivance the syringe need not be removed from the vein whilst changing these solutions. One can also thrust the needle by itself into the lumen of the vein, and only if blood flows out, attach the syringe and inject; but without a special device to prevent the needle from moving whilst in the lumen of the vein and during the attaching of the syringe, this modification cannot be recommended. When the injection is finished, the syringe is withdrawn and a sterilised dressing applied to the puncture. A great deal of detail in the technique could be considered: for instance, at what distance from the elbow the elastic bandage should be applied, whether the elastic bandage shall be removed when the needle has entered the lumen of the vein, or whilst the injection is made or when the injection is finished, or lastly, when the syringe has been withdrawn, how far the needle shall enter the lumen of the vein, how to fix the needle, once it is within the lumen of the vein, as without being fixed in position its point quite easily perforates the wall of the vein at another point than that of its entrance. To go into these and other details, to point out their advantages and disadvantages, is not within the scope of this paper.

In relation to the skin the intravenous injection is a percutaneous one. The subcutaneous injection, usually termed "hypodermic"—or to be accurate "hypodermatic"—may, on account of the drug injected, cause irritation in the connective tissue beneath the skin. That is why one has substituted for it the injection into a muscle, therefore termed "intra-muscular," or, as it is ordinarily made into the buttocks, "glutæal" injection. As in both cases the liquid is driven into the body, and to find room, has to push from one another the meshes of the connective tissue or the fibres of the muscle, whilst, in the intravenous injection, the liquid is driven into a cavity, one might conclude the latter to be less painful than the former. This theoretical consideration is borne out by practical experience. With regard to the absorption of the injected medicament—as the muscles are comparatively poor in lymphatic vessels—in the intra-muscular injection the solution is absorbed more gradually than in the hypodermic; but the absorption is of longer duration, while, in the intravenous injection, the absorptive action takes place more quickly still than in the hypodermic.

Now, how did Landerer come to use the intravenous injection? He had happened to obtain favourable results in cases of tuberculosis from balsamum peruvianum in local application; so he quite naturally considered how he could render this remedy useful for internal tuberculosis. The tubercle bacilli that cause the morbid tissue changes, he knew, are brought by the blood-stream. The same blood-stream, he argued, ought to be the means of getting the healing agent to the diseased parts of the body. In this way he commenced to try intravenous injections of an emulsion of balsamum peruvianum. Later on he discovered that the healing power was due to the cinnamylic acid which is contained in that balsam, and finally he found aqueous solutions of cinnamate of sodium, termed "hetol," the most suitable for intravenous injection.

Certain living cells, for instance the white cells of the blood, termed leucocytes, are attracted to or repelled from various chemical or bacterial irritants. This sensitiveness of the cell is known as chemotaxis—positive chemotaxis when the cell approaches the irritant, negative chemotaxis when the cell is repelled by the irritant. It is found that the chemotactic property is possessed by many pathogenic organisms as well as by certain chemical substances. The introduction of a substance with positive chemotactic property into the circulation results in the appearance in the blood of a vast army of newly-arrived leucocytes. The increased number is effected by a new formation of leucocytes in their places of production, and by an increased supply of them from all available sources.

Cinnamylic acid and its salts have high positive chemotactic properties. Two or three hours after endovenous introduction of a hetol solution, the number of leucocytes commences to increase and goes from 4,000 to 6,000 in each cubic millimetre of the blood up to 12,000 to 18,000, nay, even to 20,000, the hyperleucocytosis culminating in about eight hours, and terminating at the end of about twenty-four hours. In the healthy body this phenomenon appears and disappears without leaving any trace. If, however, there is some kind of tuberculosis in the body, the leucocytes are deposited at the diseased parts, and if one goes on injecting, more leucocytes come along, emigrating out of the capillaries of the neighbourhood. The capillaries become dilated, serous fluid transudes from them and produces œdema. As early as the third week one can see a wall of leucocytes surrounding and encapsuling the tubercle and thus separating it from the neighbourhood, and at the same time the leucocytes commence to leave the wall and immigrate into the tubercle, and provided the injections are continued, more and more leucocytes arrive at the outer layer of the peripheral wall and make it thicker and denser. The leucocytes increase rapidly in number, they seem, so to say, to inundate the tubercle, till they occupy the dominant place. In the now diminished tubercle the destructive process gradually ceases, the leucocytes change into connective tissue, and in the room of the wall of leucocytes in the periphery, and of the leucocytes within the tubercle, striæ of connective tissue appear partly of concentric, partly of radial arrangement, according to the previous shape of the trains of leucocytes. Finally, the connective tissue is shrinking, and where the morbid tissue change has been, there is now a cicatrix, where there are no more bacilli.

This is the histological process as established in a considerable number of experiments on the animal and observed in different stages on the occasion of autopsies in the lungs of individuals who had been treated with hetol. Moreover, histological investigations of tissue parts at different times taken out of the tuberculous larynx of living individuals undergoing the hetol treatment have confirmed the said change taking place during treatment. The latter investigations have been made in the private hospital of Professor Krause in Berlin, one of the medical men in attendance upon the German Emperor Frederick III.

The histological process is the same as is observed at the spontaneous healing of tubercles; but it is more lively and quicker, and as Professor Krause has shown in comparing sections out of the larynx of living individuals under and without hetol treatment, the leucocytes appear in every stage of the development of the tubercle, whereas, in the course of the natural healing, they appear but shortly before the stage of caseation. Otherwise the healing process in the course of the hetol treatment is the same as nature's healing method. In both cases we find the tubercles transformed into connective tissue in which calcareous deposit may take place.

The intravenous hetol injection is painless and harmless, and has no inconvenient after-effects. If it be followed by elevation of temperature, the dose was too high. Generally the question of dosage requires great consideration, especially as long as the process of encapsulation and separation of the tubercle is in progress. The blood-vessels in the neighbourhood of the tubercle become, as we mentioned before, dilated, and as usually their wall already is slightly affected, a rupture of some of the vessels is quite a natural thing and gives rise to the admixture of blood with the sputum. This sign is of no importance; it only shows that the treatment is really an efficacious one. If, however, an overdose is given, slight hæmorrhage may occur which—it should not be overlooked—may equally well happen without any treatment.

In uncomplicated cases of pulmonary tuberculosis the fever gradually disappears and the temperature becomes normal and remains in that state, the night sweats disappear, the appetite returns, and the weight increases, cough and expectoration diminish, bacilli and elastic fibres gradually disappear from the sputum. The septic fever met with in mixed infection is not influenced by hetol, whereas the pure tuberculous fever, as a rule, disappears at the end of two or three weeks. In this respect I may mention that a number of cases has been observed in which the patients continued to maintain a more or less high temperature, although, for months, they had the advantage of mountain air and were kept in bed, and then, under hetol treatment, the fever subsided in a few weeks. The expectoration is decreasing from the fifth or sixth week, sometimes there is a change in the character of the cough, which becomes dry, the bacilli are diminishing from the sixth week. Accordingly the physical signs alter, the area of dulness becomes smaller, the percussion note decreases in dulness, but the dulness does not disappear entirely, since the cicatrices are airless, the rhonchi gradually disappear, the bronchial character of the breath-sounds changes; where there has been bronchial breathing, one hears now weakened undeterminate sounds.

The total results of the hetol treatment of pulmonary tuberculosis are 70 per cent. cures and permanent improvements, including advanced cases. If one takes into consideration the early stages only, the results are much better. But there are very remarkable reports on advanced cases also permanently improved, and even cured, by hetol treatment. Of great interest is the case of a man who had been treated with hetol for quite a long time and died, as was thought, of tuberculosis; but the autopsy showed as cause of death degeneration of the cardiac muscle, and the cavities in the lungs were found healed with smooth thin fibrous walls—a sign that the reparative process had been established.

As a special advantage it is claimed that the patient can continue to follow his occupation during the treatment.

Of the cases of pulmonary tuberculosis I have had the opportunity of treating since coming to Australia, I could only keep in view a small percentage.

My first case was of a man 27 years old, who was suffering from phthisis for years and was frequently troubled with hæmorrhages. He had some forty intravenous hetol injections, no other medication whatever, and was permanently cured—now six years ago. Another case was of a young man of 19 years of age in a rather advanced stage. Yet for months he was improving; some weeks his weight increased by 1 lb. or 2 lb., other weeks he remained stationary, and he was getting lively, as the people with whom he was staying said, whilst before they had found him in rather low spirits. He had been an inmate of several public hospitals, had been under private medical attendance for long stretches of time, but, as he said, he never felt better and never had so much benefit from any treatment. He went away for a fortnight to visit relations, got influenza, and was admitted to a general hospital, from which he was sent to an institution for incurables; where he died in two months' time. In this case I combined hetol with tuberculin, and used internal medication as well. In some cases hetol seems to be more or less inert just like tuberculin; I then try tuberculin and use both or one of them according to the individual reaction. Two other cases I got in the very early stages became quite well, under internal medication only.

Another case, a rather interesting one, refers to a man 38 years old, who was supposed to be suffering from pulmonary tuberculosis for a long period and was for years treated accordingly by leading medical men, and finally sent to a sanatorium where he remained for four months, at the end of which, he said, he was so stout that walking was an inconvenience to him, whilst he felt no improvement whatever in his lung trouble. There were indeed all signs of pulmonary tuberculosis; the sputum examined at the Bacteriological Institute contained tubercle bacilli. Hetol injections were without apparent result. Somehow, on account of some peculiar features, I began to doubt the diagnosis. I got the sputum examined again, and the bacilli found were neither numerous nor appeared well-formed or very virulent; and finally I found out that the tuberculous process was of but a mild character and only secondary to a certain specific disease contracted about twenty years ago and established principally in the lungs. All symptoms, including the tubercle bacilli disappeared under antispecific treatment consisting in subcutaneous, intra-muscular, and intravenous injections of solutions respectively—emulsions of mercury salts. This case appears apt to support the view that the tubercle bacillus is only capable of taking root in individuals whose nutrition and power of resistance are greatly lowered, and that tuberculosis may be looked on as the sequel of another debilitating condition due to accident or disease.

There are many reports of favourable results also in tuberculosis of other organs.

Quinine hydrochloride and corrosive sublimate for intravenous injections were introduced by Baccelli, and Credé followed with Argentum Colloidale termed "Collargol." Arsenic preparations for intravenous injection were first recommended in 1897; there was sodium cacodylate, new cacodyle, then atoxyl, and finally salvarsan. Atoxyl, besides being used by itself, was combined with tuberculin and with hetol. In this connection it way be mentioned that tuberculin by itself has been used intravenously, and that for severe cases of diphtheria, Behring's serum has been recommended for the same kind of application.

Recently F. Mendel has developed the method. He commenced in 1902 with sodium cacodylate, then he used atoxyl by itself and in combination with tuberculin. Moreover, he has the credit of having introduced or rendered useful for intravenous injection three other chemical agents—fibrolysin, digitalis, and attritin.

Fibrolysin is a chemical combination of thiosinamin and sodium salicylate. Thiosinamin was known to be without effect when administered internally, but to soften scar tissue, when applied by way of subcutaneous injections. Thus it was used with favourable results in all kinds of pain caused by scar tissue and fibrous indurations, of strictures, scleroderma, adhesions, external and also of internal organs with each other and with serous membranes, of difficulty in hearing caused by cicatrices in the internal ear or fibrous adhesions of the ossicles, furthermore, in cases of chronic parametral exudates, fibrous tumours, &c. The great drawback in its application is that the drug is very difficult of solution in water, that the alcoholic solution subcutaneously applied, is very painful, and that in its solution with warm water and glycerin the drug easily precipitates. However, fibrolysin, a white crystalline powder, is easily soluble in water; but as the solutions do not keep, the drug is sold solved in ampules of amberglas. The subcutaneous injection of this solution is painful, the intravenous injection is painless, and the intra-muscular injection is practically painless. It is advisable to apply it either intravenously only or to use it intravenously for the first injection and intra-muscularly for the following ones, the latter way being best employed also with salvarsan. The indications for the application of fibrolysin correspond with the ones for the use of thiosinamin. Like thiosinamin, fibrolysin exercises a selective influence over all pathological changes—no matter of what kind of origin or in what part of the body—in which connective or fibrous tissue occupies the place of previous normal cells. The selective influence presents itself as a serous imbibition of the scar tissue, whilst normal connective or fibrous tissue is not influenced by therapeutic doses. It is solely the scar tissue that is influenced; but as this tissue may be found in any and every organ, the field for the application of fibrolysin is a limitless one. Good results have been obtained in cases of large cicatrices from burns impeding movement, indurations, and adhesions after disease, after inflammation or after injuries, sprains, contusions, fractures, traumatic epilepsy, pleurodynia after pleurisy, ankylosis, neuritis, sciatica, ulcus cruris with thick hard walls, strictures, stenoses. It is invariably the pain that induces the patient to look for help. Also in cases of pain from adhesions after abdominal sections, fibrolysin has given good results. In such and in similar cases it may render an operation unnecessary. A pylorus-stenose is not dilated by the action of fibrolysin *per se*, but the softening of the tissue surrounding the stenose enables the physiological movements of the stomach to effect dilatation. In many cases it is the normal movements of the organs respectively: muscles that can do the trick after the drug has done its work. In other cases the administration of the drug has to be followed by mechanical, hydriatic, or electrical treatment.

Intravenous injections of digitalis preparations have been performed with results that, except as to the rapidity of the action, do not appear superior to the results of internal medication. The indications ought therefore to be limited to cases in which an immediate effect seems of necessity or advantage, and in which the internal medication has no effect or is not well supported. The preparations chiefly used are digalen and strophantin. Mendel uses digitalone (Parke, Davis, & Co.).

Attritin is termed a solution of sodium salicylate in combination with caffeine. Intravenous injections of attritin have been made in rheumatic affections with excellent results. For obvious reasons they have been resorted to, and are still resorted to, mostly when internal and external medication have failed. In these cases as well attritin is effective and besides is devoid of all unpleasant appearances connected with internal medication of salicylates. Pain and exudations on rheumatic basis disappear with certainty and rapidity. On the other hand the effect is a specific one as obtained only in rheumatic cases—which fact having abundantly been confirmed, is of the utmost importance for the differential diagnostic. There are not a few cases in which the diagnosis rests between tuberculosis and rheumatism, in some chronic joint affections it is quite impossible to make the distinction. *One* injection settles the matter. In the very first case of rheumatism I treated intravenously—it was at the commencement of 1907 at Mount Morgan—I had a very good result; it was an elderly man crippled from chronic joint-rheumatism who could not walk nor move his arms. Each injection relieved the pain and enabled him to move more or less one or the other limb. At last he could move his arms quite freely and could walk fairly well—fairly well, as he had had one leg fractured. This success brought me a number of patients. Some were cured by one or two or more injections. Some asked me to try, although I anticipated inefficacy; the ones who, in spite of my doubts, remained convinced they were suffering from rheumatism, were disappointed. Never shall I forget a young woman who for years was suffering, as she firmly believed, from rheumatism of the hip, and had undergone many courses of treatment, had been at Rotorua, &c., and was very hopeful on account of what she had heard regarding the effect of my injections. The first injection was without any effect. I made another one—again without effect. I told her I doubted her rheumatism. She become very disheartened and commenced weeping. At her request I made a third injection, once more without effect. I told her then I did not consider her ailment rheumatism at all, and would definitely abandon the injections. Thereupon the woman became quite hysterical and could scarcely be consoled. Afterwards I learned she had a suppurative discharge from the vagina.

As the principal advantages of the intravenous injection we can put forward smallness of the dose necessary, complete absorption, painlessness, certainty and rapidity of action. Hetol occupies an exceptional position, inasmuch as the action is slow and gradual, and a cure requires comparatively a long period. The rapidity of action appeared to me most striking in the injections of attritin, corrosive sublimate, and digitalone. In a case of valvular heart disease I injected (having not yet digalen or strophantin) digitalone, because the patient was sick of continually taking medicine; she told me that one injection put her right for two or three days, just as one 8-ounce bottle of her usual medicine containing infus. digital., tinct. digital., tinct. strophanti, tinct. scillæ, &c. From the intravenous injection of corrosive sublimate I did not see any effect that could not have been obtained from subcutaneous or intra-muscular injections of mercury salts as well. Attritin injections have given me very good results, better results that any other treatment, also in chronic cases, as long as the character of the originally rheumatic affection was unaltered.

As the essential pathological changes in chronic rheumatism, we find white fibrous tissue within the structure of the articulations, tendons and their sheaths, aponeurosis, periosteum, septa and sheaths of muscles, nerves, &c. The process that causes the lesions consists in an inflammation leading to indurations of the connective tissue. The induration may involve a more or less considerable portion of a nerve or muscle-sheath, or of an aponeurosis, but even a whole aponeurosis may become indurated, or a cord or strand of thickened fibrous material may run through its entire length. This is apparently the same pathological condition so often witnessed after fractures, sprains, contusions, which we have mentioned under fibrolysin. Here and there we had occasion to refer to sciatica as suitable for the respective injection, the final stage of pathological condition from rheumatism being the same as the one after injuries. The identity of the condition in the final stage appears striking in considering a well-known and extensively found ailment—lumbago. This affection is generally regarded as of the nature of muscular rheumatism, but by some as the result of fibrositis. Whether in the case of original rheumatism some of its poison be left in the fibrous tissue, remains to be proved. Anyhow, in the majority of cases the etiology is obscure, and one has to chance attritin or fibrolysin. Most of the cases require repeated injections, but not necessarily of the same drug. If there be no effect from attritin, one ought to try fibrolysin, and *vice versâ.* If there be an effect, however slight, one ought to keep on injecting the same drug. Some patients who had heard of cures of mine by one injection, but had no benefit from a single one, did not come back. An injection of the other drug, or even an injection of the same drug, might have been beneficial. Cases like these suggest trying to combine the two drugs in the same injection.

Permanent cures from one injection to which I have just referred, comprise one case of pleurodynia and some cases of lumbago. The former was of a man of 37 years of age, who for years had been suffering intense pain, frequently recurring, sometimes with increased severity, in the side of the thorax, and who had unsuccessfully been treated by five or six medical men, none of whom failed to hand him a prescription. In one case of lumbago the patient came into my consulting room limping on crutches. I made him sit down in an easy position and made an injection. About two minutes afterwards I told him to get up. He did so, but was exceedingly careful in doing so, fully expecting to experience the too well known violent pain. But the pain was gone, and scarcely had he straightened his back than he exclaimed: "Marvellous!" He left my consulting room quite straight. Finally, I may mention the case of an elderly lady feeling much discomfort and inconvenience from lumbago. I found her resting in bed—or rather restless, as, now and again crying with pain, she was continually moving seeking for an easier posture. She had immediate relief from the injection I made. I did not see her again nor did I hear anything of her. By the way, she was not hysterical or neurasthenic. Patients of this kind nowhere fail to put in an appearance.

In conclusion, we cannot help reminding our readers that there is not a tittle of justification to condemn the method on account of its dangers. As we pointed out before, all risk can easily be eliminated if the necessary precautions are taken. And if unpleasant incidents have occurred, they could be accounted for by some error or oversight. The only unpleasant incident I have met with was in the case of a man 70 years old, who was attacked by a tremor all over the body; he said he felt like fainting, but he did not faint, rather soon recovered. In this instance the solution was prepared and sterilised in a Friendly Societies' dispensary, and I used it as it was brought to me from the dispensary by the patient. In fair justice I may state that I have had the same formula frequently prepared and sterilised in the Mount Morgan Friendly Societies' dispensary and used the solution just as it came from it, without ever having experienced any unpleasant symptom; of course, once the bottle was opened, I myself sterilised whatever I used from the contents. Since that incident, however, I have made it my custom to prepare and sterilise the solutions myself.

Another unpleasant incident is said to be pain and tension all over the vein. My own experience in that respect is that the injection causes pain and tension—more correctly pain by tension—if the vein had been rendered rather turgid and considerable pressure is required for the injection. Immediate relief is brought about about by loosing the bandage and thus allowing the blood with the injected solution to flow into the general circulation. It is not necessary, not even advisable, to loosen the bandage suddenly, as can be done by using a pneumatic bandage constructed in the manner of the Riva-Rocca bandage.

As regards measures best adapted for the prevention of tension and pain—the vein should not be rendered turgid, it is sufficient to render it prominent; of course, the more prominent the vein is, the easier is the injection. Then the site of the injection should not be too near the bandage; that means the bandage ought to be applied as far from the elbow as possible, especially if a comparatively large quantity of fluid is to be injected. Thirdly and lastly, as the capacity of the vein is a limited one, it is important to manage in that way that the use of rather large quantities is avoided, and only small quantities are injected.

In the latter respect the hetol injections occupy a very favourable position. Hetol solutions can be prepared or obtained in glass tubes in 1, 2, and 5 per cent. strength. A commencement is made with 1 milligram hetol, for which dose naturally the 1 per cent. solution is used. The dose is gradually increased by ½ to 1 milligram at each subsequent injection up to 25 milligrams. Thus it is easily seen that the different strength enables one to inject small quantities of fluid, however high the dose may be. The hetol injections are made every second day or three times a week. Landerer emphasises the exclusive use of the chemically pure sodium cinnamate synthetically prepared by Messrs. Kalle & Co., Chemical Works at Biebrich o/Rhein.

The solution for the treatment of rheumatism contains 70 grains of soda salicyl. and 10 grains of caffeine in an ounce of distilled water. Two cubic centimeter of this solution are injected. Digitalone (Parke, Davis, & Co.) is used in the same dose. Fibrolysin is supplied in ampules containing 2·3 c.cm. of fluid. This is the largest amount of fluid generally used. With the introduction of salvarsan, larger quantities have to be injected. For this event I think it a good plan to inject in several portions and to loosen the bandage in the intervals.

The good, nay excellent, results obtained with the intravenous injection, put this application of drugs on the level of a good method capable of development, and ought to stimulate attempts to improve it as regards the technique and as regards drugs and combination of drugs especially suitable. One is pretty safe in asserting that the intra-venous injection will never become the generally adopted method of administering drugs. But already now the method has proved extraordinarily useful and beneficial, and the field for its application deserves and probably will be enlarged in the future. And if some authors consider and speak of the intravenous injection as innocent as an inunction, we quite agree with them—*cum grano salis*.

II.

SECTION OF SURGERY.

PRESIDENT'S ADDRESS.

By G. A. Syme, M.S. (Melb.), F.R.C.S. (Eng.).

(Surgeon, Melbourne Hospital ; Lecturer and Examiner in Clinical Surgery, Melbourne University; Consulting Surgeon, St. Vincent's and Queen Victoria Hospitals.)

The introductory words of most presidential addresses at Congresses are so similar that almost they might be stereotyped. The speaker expresses his appreciation of, and gratitude for, the honour conferred, and his deep sense of his own unworthiness for the position. The phrases are not mere formulæ, demanded by custom, but express the sincere feelings of each successive occupant of the chair, as most certainly they do my own. To one like myself, whose life has been spent in the study, teaching, and practice of Surgery, no greater compliment could be paid than to be invited to preside over this section. I feel it an especial honour to occupy this position at such a notable meeting, held in this beautiful Mother City of Australia, which is so splendidly equipped with magnificent hospitals, and where the best traditions and highest attainments of our surgical art are so admirably exemplified. I am exceedingly grateful.

Since this Congress met last in Sydney in 1892, the progress of surgery has been admittedly very great. If, however, you will read, in the transactions of that meeting, the very able and memorable address by the late Dr. Wm. Gardner, as President of this section, you will, I think, agree that the advance has not been quite so remarkable as some are apt to think, especially when they see sensationally-headed paragraphs in the lay press about the marvels of modern surgery. Let me recall, very briefly, some of the chief subjects dealt with by Dr. Gardner, in his presidential address, nearly twenty years ago. He showed that aseptic technique, in contradistinction to Listerian methods, was firmly established. In abdominal surgery he referred to the layer method of suturing the parietes and the necessity for preventing adhesions; to pylorectomy, by Kocher's method, to gastro-enterostomy, to Loreta's operation and pyloroplasty, to resection of the stomach wall for ulcer and for gastric dilatation; to the treatment of intestinal obstruction by early laparotomy; and to resection of the cæcum and ileum when affected by tubercle. Dr. Gardner reminded us that he had performed the first cholecystostomy in Australia, and he discussed cholecystectomy, choledochotomy, and cholecystenterostomy. He also mentioned his pioneer operations on the kidney, having performed with great success practically all the operations now done on that organ. He advocated early laparotomy in appendicitis and in septic peritonitis. Reference was also made to the substitution of inguinal for lumbar colostomy; to Kraske's method of excising the rectum; and to hysterectomy. In the surgery of the brain and spine allusion was made to the work of Macewen and Horsley. He concluded by speaking of total extirpation of the larynx, which he had successfully performed.

In reviewing the surgical work of the intervening period, it will be found that, although only a few new regions have been assailed, and very few entirely new methods of surgical attack have been devised, great progress has been made in securing the safety and success of operations, and that there has been an enormous increase in the number performed. While all the procedures mentioned by Dr. Gardner were performed twenty years ago, they were done infrequently, by no means always successfully, and almost solely by acknowledged surgical masters. Now they are everyday occurrences, performed by practitioners not only at the world's centres, but at its very outposts; and with almost uniform success. As a result of improvements in the details of technique, and with extended experience, surgeons have demonstrated the almost absolute safety of operations *per se*. Want of success has been found to be due to the operation having been undertaken too late. Consequently, surgeons have been induced to operate earlier, and for purposes of exploration, and in this way has been gradually evolved a "pathology of the living," as Moynihan has termed it. By examinations made during operations surgeons have elucidated the relationship of previously obscure medical symptoms, often regarded as functional, to the early stages of morbid processes, of which the pathologist saw, *post mortem*, only the disastrous end results. Early operation disclosed not only the nature of the morbid condition, but also that it could be relieved by surgical measures alone. The consequence was a further increase in what has been called "prophylactic surgery," and a further widening of the field of investigation. Treatment of conditions formerly regarded as purely medical became more and more surgical, and so practitioners generally have been forced to become surgeons. We live in a democratic age. In all departments we find that what were once the privileges of the few are becoming the common property of many—surgery is no exception.

Side by side, however, with this wide diffusion and generalisation of surgical practice has developed a very definite specialism, and it is to those who confine their attention more particularly to intensive study in special departments of surgery that we are indebted for most of the advance that has taken place. It will be noticeable, further, that recent surgical progress has been very largely influenced by physiology, which has made such enormous strides in the last two decades. It is, indeed, claimed by some, perhaps a little over-anxious to turn an effective phrase, that we are now at the dawn of a new era in surgery, which will be known as that of "Physiologic Surgery" (I need hardly say the coiner of the phrase is an American). Whether such a view will prove correct time alone will show. There is, I think, some justification for it, as I propose to try to demonstrate in the course of this address.

One example, that immediately presents itself, of the satisfactory results of the application of physiology to surgery, and also a typical example of the best kind of specialism, is seen in the surgery of the nervous system. When travelling abroad recently it seemed to me that some of the most striking instances of the surgical progress during the last five years was to be found in this department. It was my privilege to see something of three men to whose original work much of the advance is due. I refer to Sir Wm. Macewen, Sir Victor Horsley, and Dr. Harvey Cushing. Each is a true specialist of the highest type, who, by intensive study and research, has made himself absolute master of every aspect and detail of his subject. I met Macewen at the International Medical Congress at Buda Pesth, where I heard him give an address on cerebral surgery, and also at the B.M.A. meeting at Belfast. Unfortunately, I did not have an opportunity of seeing him operate. Of the work of Horsley and Cushing I saw a good deal. It was not only intensely interesting, but highly educational. I was particularly impressed by their physiological attitude and the extent of their laboratory research. In fact, their contributions to physiology are almost as great as to surgery. The results of such specialism speak for themselves. Less than ten years ago Von Bergmann, then regarded as a high authority on cerebral surgery, wrote that operation for cerebral tumor should be limited to those situated in the motor region, or adjoining areas, that the regional symptoms corresponding to irritation and subsequent destruction of portions of the motor region must be distinctly developed before operation is undertaken, and that only a very small number of cerebral tumours can be reached by the surgeon's knife. The results of operations under such limitations were necessarily disastrous, in most cases. The growth had probably spread so widely by the time motor symptoms were the localising indications, that either it was inoperable, or the destructive effects of the lesion and its removal left the patient in a deplorable condition, owing to the loss of function entailed. Occasionally one got a completely satisfactory result, as in a case I exhibited at the last meeting of this Congress, at Melbourne, in which I had removed fifteen years previously, a dural sarcoma, which had simply pressed on the motor cortex without destroying it; or, as in cases of cerebral hydatid, in which successful results have been recorded by several Australian surgeons. So late as 1906, Allen Starr's statistics gave a distinctly unfavourable account of surgical possibilities in cerebral tumour. He stated that only 10 per cent. were open to surgical treatment, and out of 100 cases one might expect success in five. But during the last five years great advances have been made. Increased knowledge of the physiology of the nervous system, and physiological methods of investigating alterations in its functions, have enabled lesions to be diagnosed, and what is of great importance, diagnosed at an early stage, in other parts of the brain than those adjoining the Rolandic fissure; while improved technique has enabled the expert to remove them, even from the very base and sella turcica, not only with a lessened mortality, but without such destructive effects as formerly. Horsley's results are well known, though I have not been able to find any published statistics. I believe they are being compiled. Cushing reported recently that in eighteen months he had operated on 100 cases of cerebral tumour, with eight deaths in the first fifty, and three in the second fifty; with thirty complete cures, with palliation in sixty-seven cases, and with no post-operative complications in any of the non-fatal cases.

It is not only in intra-cranial conditions that progress has been made. In lesions of the spinal cord, especially growths not involving the cord itself, and of the nerves, physiological methods have led to surgical success. The work of Dr. Basil Kilvington, of Melbourne, is an excellent illustration of physiological surgery applied to the nerves, and it has been widely recognised as of the highest order, as well as practically useful.

Another field in which physiological research, specialist intensiveness, and surgical progress have gone hand in hand is disease of the thyroid gland. Here, again, Victor Horsley was a pioneer, not as a surgeon, but as a physiologist; while any mention of the surgery of the thyroid suggests at once, almost subconsciously, the revered name of Theodor Kocher. His masterly address on the surgical treatment of Graves' disease at the B.M.A. meeting, in London, must be still fresh in your memories. He not only placed before the meeting the results of his so far unequalled surgical experience in this disease, but showed a profound knowledge of physiological research, and gave evidence of much original investigation. I say that Kocher's experience in operating is unequalled so far, advisedly, for Dr. C. H. Mayo must by now have operated on nearly as many cases as Kocher, and if Dr. T. P. Dunhill, of Melbourne, continues as he has begun, he will soon equal them both in operative experience. The story of surgical success in Graves' disease is similar to that in the case of the nervous system. Physiological and pathological research elucidated the essential cause of the symptoms, and suggested surgical treatment. The results of operation confirmed the view that hypersecretion was a main factor in producing the condition, and extended experience has shown that early operation is both safe and satisfactory. In this connection I would like to remind you of a remarkable paper contributed by the late Dr. J. O. Closs to this Congress, at its session in Dunedin, in February, 1896—a paper which has never received the attention it merited. Dr. Closs reported several cases of Graves' disease, in which he had most successfully performed thyroidectomy. He "urged the physician to early recognise that Graves' disease is one for surgical treatment, and not to delay till his therapeutics have been exhausted, and the case become so bad that both surgeon and patient are handicapped in a contest that can, in all probability, have but one ending." The remarkable feature in Dr. Dunhill's results is that he claims to have operated on most advanced and degenerated cases, not only with practically no immediate mortality, but with a permanent cure of the general condition and secondary organic changes. Kocher stated, "The more I have seen of the disease the more I am convinced that the continuous poisoning of the system with thyroid secretion brings on in long-standing cases organic changes in the heart muscle, in the kidneys, in the liver, in the pancreas, and in the cells of the cortex cerebri," and consequently operation "should only be done after most careful consideration." C. H. Mayo says that in all his cases that died "there was degeneration of heart muscle, fatty liver, soft spleen, chronic nephritis, and usually enlarged thymus. The various degenerative changes have explained why in advanced cases the mortality is not only increased, but also why a satisfactory cure does not follow in many instances where great operative skill has avoided mortality. Such cases should be compared to the removal of a bullet from the body with the expectation of obtaining relief from all injury caused by its passage." In the light of these opinions it is somewhat difficult to understand Dr. Dunhill's successful results in advanced and degenerated cases.

It must be remembered, however, that Kocher stated, also, that "even in cases where we found irregular heart action, glycosuria and albuminuria, we have seen a complete *restitutio ad integrum* when we had succeeded in avoiding the immediate post-operative dangers."

These undoubtedly can be avoided by using local anæsthesia, and by skilful, rapid, and, above all, gentle operating. I have had the privilege of seeing thyroidectomy performed by C. H. Mayo, at Rochester, by Von Eiselsberg, at Vienna, by James Berry, in London, and by Dunhill, in Melbourne, and the last is unquestionably exceptionally gentle and skilful. Judgment has to be exercised, also, in determining the amount of thyroid to be removed, and apparent failure may be due to insufficient removal. Skill and discrimination in operation will account for Dunhill's immediate success, but do not altogether explain the cure of the secondary degenerative changes. Must we assume that the poisonous effects of excessive thyroid secretion on the different organs are not permanent, and if the poison is removed the organs can recover? The point to be emphasised, however, is that these degenerative changes should not be allowed to occur, but, as Dr. Closs urged fifteen years ago, should be prevented by early operation. It does not follow that every case of hyperthyreosis demands operation; as Kocher observes, "when a larger amount of thyroid secretion is needed by the healthy organism, as it is at puberty, in pregnancy, and in some diseases, hypertrophic swelling, with hyper-activity of the gland, is a useful physiological process, and it is a great mistake to interfere by operation, as is sometimes done." I have personally seen several such cases, which have completely recovered without the operative treatment which had been advised. Associated with many cases of Graves' disease, even in an early stage, is the status lymphaticus, and when fatalities have occurred during or soon after operation, this complication has generally been present. If recognised, it should be regarded as a distinct contra-indication to operation under a general anæsthetic; whether it is a bar to operation under local anæsthesia may be debatable. I should not care about operating myself if sure the status lymphaticus was present.

Physiological experiments have demonstrated the function of the parathyroids to some extent, and occasionally, fortunately rarely, the danger of removing them when operating on the thyroid has been proved to be very real in practice. The experiences related by Dr. W. H. Brown, of Colac, to the Victorian Branch of the British Medical Association last year, were of extreme interest, but no one would care about repeating them. After a second and extensive removal of thyroid gland, for Graves' disease, tetany developed. After many trials of parathyroid extract, and implantation of the parathyroids and thyroid of animals, the condition was cured by implanting human thyroid and parathyroid removed immediately after death. The grafting of thyroid and parathyroid tissue has hardly passed the experimental stage, and its full value is not yet determined. It seems to be essential that the graft be homologous, and advisable to attach its blood vessels to those of the patient by arteriorrhaphy.

The surgery of the thorax and its contents has been greatly advanced in recent years by experiments in the physiological laboratory, resulting in the introduction of means to prevent dangerous collapse of the lungs, and acute pneumothorax. My own experience, admittedly limited, of operations on the human thorax, when only one pleura was opened, has inclined me to think that the danger of pulmonary collapse has been overestimated. In dealing with unruptured hydatid I have frequently opened freely into the healthy unadherent pleura, and never noticed any untoward symptoms. In August, 1900, at the Medical Society of Victoria, I showed a case in which I performed a most extensive operation through the thorax for the removal of a sarcoma involving the parietal and pulmonary pleura, the lung, the diaphragm, and the spleen. The shock was severe, but there was no marked collapse of lung, and the pericardium being fully exposed, it could be seen that no appreciable displacement of the heart occurred. I have also not infrequently accidentally opened the pleura, and have never seen any unpleasant consequences. That they do not always or even usually result is, in fact, well known, and was long since pointed out by Matas, Trendelenberg, and others. All the same, calamities have occurred, and we are not warranted in disregarding the methods now available for preventing them by the use of some form of differential pressure chamber, or by the intratracheal insufflation method, devised by Meltzer and Auer. At the August meeting of the Victorian Branch of the British Medical Association, this year, I showed a patient from whom I had removed an extensive sarcoma of the thorax, using intratracheal insufflation. The method was entirely successful, keeping the lung well inflated; and no difficulties were experienced, although the apparatus was only an extemporised one. The practical results of very extensive intra-thoracic operations on the lungs and œsophagus in the human subject with differential pressure apparatus have not been very encouraging so far. Dr. Willy Meyer's experiences, while testifying to his courage and perseverance, seem rather a record of failure and fatality.

In the surgery of the vascular system almost phenomenal progress has been made. It has been pithily remarked that "The road to the heart is only 2 to 3 cm. in a direct line, but it has taken surgery nearly 2,400 years to travel it." The successive steps on the road have been, as in the case of other organs, pathological anatomy, experimental physiological research, and operative experience, which practically began in 1896. The most recent statistics of operation for wounds of the heart that I know of are of those of Brewster and Robinson, in the Annals of Surgery for March, 1911, giving 177 cases with 71 recoveries. The essentials for success are prompt diagnosis and immediate operation, with absolute asepsis. No uniform method of approach has been adopted, but if the lung can be kept inflated by differential pressure of some kind, an intercostal and transpleural approach will probably be found to be best.

Arteriorrhaphy has now been so perfected as to be preferable, in many circumstances, to ligature, and here again laboratory research paved the way for operative success. At a meeting of the Medical Society of Victoria in February, 1904, I showed a patient whose torn brachial artery I had successfully sutured. The method used was approximation of the cut ends by a continuous suture of very fine silk. The case was complicated by dislocation of the elbow and much bruising, and according to Matas, suture of the artery is contra-indicated in such circumstances. I tried it, because the vein was also torn and the artery divided above the anastomotic branches, so that I thought there was risk of gangrene after complete occlusion of the vessel. The technique elaborated by Carrel is undoubtedly the best for arteriorrhaphy, and by it he has performed on animals most astounding operations. He has successfully united the abdominal aorta and vena cava and reversed the circulation; grafted a segment of the vena cava into the aorta; and transplanted the heart of one animal to the neck of another. Carrel's papers on the experimental surgery of the aorta and heart read like romance, but he is careful to say that such operations must not be performed on human beings.

The methods devised by Matas for the treatment of aneurisms, directly, by endo-aneurismorraphy, are almost revolutionary, yet it cannot be denied that they are scientific, conservative, and practical. The Hunterian method of ligature is so classical, and generally satisfactory, that English surgeons have been slow to try any other, but the results attained by Matas and his American confrères are forcing them to abandon their conservative attitude. In a case of arterio-venous aneurism, due to bullet wound, which was shown at the Medical Society of Victoria, in March, 1900, I laid open the sac, and closed the openings of the vessels into it by endo-saccular suture. The result was so satisfactory that I determined to perform endo-aneurismorrhaphy on the first suitable pathological aneurism that came to me for treatment. Notwithstanding the alleged prevalence of syphilis in Victoria, surgical pathological aneurisms appear to be much rarer than they used to be. When I was a student and resident medical officer at the Melbourne Hospital, I remember seeing several cases treated by digital compression and ligature, but in the last seven years only two cases have been treated there, both by ligature. It was not until quite recently that an opportunity occurred of operating by Matas' method. On August 11th, of this year, I did an obliterative endo-aneurismorraphy on a popliteal aneurism. The efficiency of the collateral circulation was previously tested by the plan recently suggested by Matas, of making the limb ex-sanguine by an Esmarch bandage and an old-fashioned pad tourniquet, removing the Esmarch and noting the returning blush in the limb below the tourniquet. This proved quite satisfactory, and the operation itself was simple and successful.

Abdominal surgery has continued to make steady progress since we met here twenty years ago. Dr. Gardner did not refer to either the spleen or the pancreas as objects of surgical interference, although hydatid of the spleen must have been treated by Australian surgeons before that time. My own experience antedated it by five months, as I operated on a hydatid of the spleen on 1st April, 1892, by the transthoracic route, excising a portion of the tenth rib and incising the diaphragm. The cyst was marsupialised and drained, as it was suppurating. All subsequent cases of splenic hydatid I have dealt with by a muscle-splitting abdominal incision, removal of the mother cyst, and closure of the incision, without drainage. Physiologists have not thrown much fresh light on the functions of the spleen. It seems extraordinary that such a large, vascular and single organ can be removed, and may be congenitally absent, without the health being affected to any appreciable extent. On the other hand, in Banti's disease the pathological condition of the spleen seems to be the cause of the symptoms, which are greatly improved by the removal of the enlarged and diseased organ. My colleague, Dr. R. A. Stirling, has removed the spleen in six cases with great success. My own experience is limited to three cases, all typical, and fairly advanced, which have remained perfectly well after splenectomy. I have also recorded a somewhat unusual case, in which I successfully removed a very movable wandering spleen, which had become rotated on its pedicle.

The surgery of the pancreas has been evolved from that of the bile passages. The association of inflammation of the pancreas with gall-stones was brought under my own notice nearly twenty years ago by the first case in which I performed choledochotomy, for a stone at the lower end of the common duct. At the autopsy a suppurative pancreatitis was found, evidently existent before operation. On anatomical grounds such an association seemed very natural, and I was surprised how little information I could get on the subject of pancreatitis, and especially in relation to gall-stones, in the literature then available. The classical papers of Fitz were published in 1889, but it was not till 1900 that references to surgical diseases of the pancreas began to appear in medical periodicals. Foremost among the surgical pioneers was Mayo Robson. In the introduction to the last edition of his monograph on "The Pancreas, its Surgery and Pathology," he lays especial emphasis on the fact that our knowledge has been gained chiefly by a combination of physiological research with investigations made during life at abdominal operations. One cannot add anything to what is to be found in Robson's book, but perhaps I may be permitted to record some very limited personal experiences of pancreatic surgery. I have operated on three cases of acute pancreatitis. In two I simply opened the upper abdomen and drained. In the third case I also incised the pancreas. All recovered. One patient was shown at the Melbourne meeting of this Congress, the second case remains well, the third died twelve months after operation, from some other cause, with no obvious symptoms of pancreatitis in the interval. The post mortem examination revealed that the pancreas was enormously enlarged, but hardly any normal pancreatic tissue remained, its place being taken by fibroid tissue and spaces full of altered blood. In this case, and also in the others, I was puzzled to understand how the operation beneficially affected the condition. The patients all seemed desperately ill, and apparently were restored to health after the operation. It may be said that they would have recovered if left alone, but I can hardly think so. Several similar cases have been recorded, and it has been suggested that the relief of tension and drainage prevents absorption of toxins, and arrests the progress of peritonitis.

Of pancreatic cysts I have seen four examples. Three were ordinary retention cysts, which made a tedious recovery after marsupialisation and drainage. The fourth is still under observation and is of a doubtful nature. It was thought to be a hydatid of the left lobe of the liver, but at operation proved to be a cyst in the body of the pancreas, and the adjacent portions of the gland, forming its walls, were also studded with cysts of various sizes. An attempt was made to resect the cystic area, but had to be abandoned, owing to hæmorrhage and the density of the adhesions. A considerable portion of the cyst wall was removed, and the rest marsupialised and drained. The main cyst gradually contracted and eventually closed, but the track re-opened and discharged glairy fluid, presumably from some of the remaining smaller cysts. Robson states that "if several small ducts are constricted, the resulting cysts may be small and multiple." In this case an examination of the fluid at the physiological laboratory of the Melbourne University showed that it did not contain any digestive ferments, so that the cysts were not probably due to retention. Microscopically, the cyst walls were lined with cylindrical epithelium, with no evidence of carcinoma. The condition was presumably cystic adenoma, or congenital cystic disease, which is very rare.

Mayo Robson has recently suggested that surgery may do something of a prophylactic nature in cases of diabetes. He is of opinion that diabetes may be avoided by the early recognition of interstitial pancreatitis, and its treatment by draining the bile ducts and so indirectly the pancreatic ducts, at the same time surgically removing the cause, whether that be gall-stones, duodenal ulcer, or other conditions. It may seem presumptuous to venture any criticism of the views of so eminent an authority, and one whom everyone respects so highly, but I must confess that his reasoning on this particular question does not appear convincing. He admits that to cause diabetes the islets of Langerhans must be affected, and that when this is caused by the inter-acinous form of pancreatitis surgery can do nothing. Further, that in the pancreatitis that is secondary to gall-stones, the affection is interlobular, not affecting the islets. In some cases, however, glycosuria may be associated with a pancreatitis secondary to, or at least also associated with, gall-stones. To explain such cases and the possibility of benefit from operation, he assumes that the inflammation of the pancreas while of the catarrhal type acts "by inhibiting the functions of the islets." The assumption is ingenious, but the evidence on which it is based seems to me insufficient. It rests on several cases of pancreatitis associated with glycosuria, in which, after operation, both the sugar and the Cammidge crystals coincidently disappeared from the urine. But I think most of us have seen cases of gall-stones where temporary glycosuria occurred, and disappeared without operation; certainly I have myself. However, the assumption may be correct; and if so, early operation is clearly indicated in all cases of gall-stones, and the digestive troubles they induce, not only for their relief, but as a prophylactic measure against still more serious disease.

Twenty years ago it would have seemed a strange proposition to discuss the surgery of indigestion. To-day the results of operative experience have proved that a majority of patients with symptoms of so-called dyspepsia have an organic disease, curable by surgical interference alone, and often only to be diagnosed accurately on the operation table. The cause of the symptoms may be in the digestive organs themselves, or affect them reflexly. One of the commonest causes of dyspepsia is gall-stones. The profession has not yet learned to regard this morbid condition as the very serious affection it really is, or to recognise that the only treatment is by operation, and by operation early, as soon as the diagnosis can be made. William Mayo has recently emphasised this opinion in a paper entitled "Innocent Gall Stones a Myth," but it is a view long maintained by Robson, Moynihan, and others, and one I strongly advocated in a paper read in December, 1896. The dangers of operation arise from complications consequent on long continuance of the trouble, and I know no disease in which it is so difficult to predict what conditions may be found at operation, when symptoms have existed for some time, and whether the operative procedures will be simple, or most difficult. Perhaps it may be of interest to refer to one unusual complication, met with recently, which was a cause of great personal anxiety. The patient had suppurative cholecystitis, with gall stones. Dense adhesions matted the gall bladder and ducts to the surrounding structures, but after a great deal of trouble, what appeared to be the cystic duct was exposed and clamped. Difficulty was experienced in freeing the gall bladder from below, so it was dissected down from the fundus, and removed. It was then found that the clamp had been really on the common duct, which was now divided, and the cystic duct and a small part of the hepatic duct had been removed with the gall-bladder, on which they were coiled up, densely adherent to its wall, while a gap was left between the cut ends of the hepatic and common ducts. With much difficulty I succeeded in suturing the ends together, but being very doubtful if they would unite, owing to the tension, a tube was passed down to the union. Through this bile escaped profusely for weeks, while the motions were absolutely without bile. Eventually, however, the motions began to be coloured, and the biliary fistula gradually closed. I am afraid the cicatrix is contracting, as the patient has had some jaundice lately. In view of the possibility of the defect in the ducts remaining permanently, I made some experiments to see whether a piece of vein could be grafted into the gap. I am much indebted to Professor Osborne for enabling me to have these investigations carried out, under his supervision, at the Physiological Laboratory, Melbourne. I succeeded in suturing a piece of the jugular vein between the hepatic and common ducts, first in a goat, and then in a large dog, but the success was not permanent, as the animals died. The union was satisfactory. There was no leakage of bile, no jaundice, and no peritonitis. Why exactly death occurred was not very clear; the animals would not eat, got feeble, and seemed to die of exhaustion. Professor Osborne informed me that interference with the biliary apparatus in the lower animals always causes great shock and general disturbance.

Another very frequent cause of symptoms of indigestion is ulceration of the stomach or duodenum. Opinion is still somewhat divided as to the best surgical procedure in such cases, and the last chapter on the subject has yet to be written. Many regard gastro-jejunostomy as *the* treatment for these conditions. In a very able paper, contributed to the New South Wales Branch of the British Medical Association in December last, Dr. Flynn gave a very clear demonstration of the close relationship between physiological and surgical progress in affections of the digestive organs. He also suggested a very reasonable physiological explanation of the *modus operandi* of gastro-jejunostomy. He attributed its good effect to the breaking of the vicious circle which exists so long as spasm of the pylorus induces excessive secretion, and increased acidity keeps the pylorus closed. The new outlet not only permits the timely exit of the stomach contents before they have become excessively acid, but also allows acid chyme to enter the duodenum, where, by the formation of secretin, it stimulates the flow of biliary and pancreatic secretions, thereby relaxing the pyloric spasm. Further experience will, I think, probably show that gastro-enterostomy will not prove so satisfactory a method of treatment, as is now believed, except where there is actual stenosis. Even then it will not always prevent the progress of ulceration, or the occurrence of hæmorrhage and other complications, as I have found in several instances in my own limited experience. I do not wish to weary you with the recital of cases, but permit me briefly to refer to one, of some interest in this connection, which came under my observation last February. A gastro-jejunostomy had been performed for pyloric ulcer some months previously. A portion of the ulcer had been excised for microscopical examination, which showed it to be non-malignant. The patient's symptoms were not relieved, but got worse. He developed jaundice and vomiting, and lost flesh. It was thought the condition must be malignant. At a second operation I found dense adhesions between the pylorus and the parietes, and binding down and obstructing the common duct and duodenum. In separating these the ulcer gave way, presumably where excised. I sutured the perforation, freed adhesions, and did a cholecystenterostomy—a drain tube was put in, through which, a few days later, gastric contents flowed freely. Here clearly all the stomach contents did not escape by the gastro-enterostomy opening, but passed over the ulcer, which had not healed, and the patient was not relieved. The correct treatment would have been pylorectomy, but I thought the patient too low to stand such an operation, especially considering the density and extent of the adhesions.

What has been termed "Appendical Dyspepsia" is another question that requires further investigation. I do not think we are at present in a position to dogmatise concerning it. My own experience indicates that chronic appendicitis undoubtedly can cause symptoms of indigestion, closely resembling those of gastric or duodenal ulcer, but it also produces symptoms and signs of its own, by which its presence can be recognised clinically, if properly investigated. I am sceptical, however, whether the classical symptoms of gastric ulcer, including hæmatemesis, can be produced by appendicitis alone, when the appendix gives no clinical evidence of being diseased. I do not think, even when the appendix is diseased, that it can be regarded as the sole cause of the gastric symptoms, simply because the exterior of the stomach when explored shows no indication of ulcer. Even when the stomach is opened an ulcer or erosion may be very easily overlooked. There is no reason why chronic appendicitis and gastric ulcer should not co-exist, the appendicitis may even be causally related to the ulcer, by exciting pyloric spasm. Since making it a rule always to open the abdomen in such a manner that it can be thoroughly explored, as I saw done at the Mayo's clinic, and by Howard Kelly, I have been surprised at the frequency with which I have found an association of morbid conditions, especially in connection with chronic appendicitis. I have at the one operation not only removed the appendix, supposed to be the chief offender, but also performed cholecystostomy for gall-stones, and gastro-jejunostomy for definite duodenal ulcer. Further, I have operated on two patients whose symptoms indicated gastric or duodenal ulceration. No external evidence of such was found, while in each the appendix was very obviously diseased, and was removed. The symptoms indicating ulcer have since returned.

A problem that waits for solution is the surgical treatment of general visceroptosis. Movable kidneys may be fixed, gastroptosis may be improved by shortening the small omentum, and ptosis of the transverse colon may be suspended. Mr. Arbuthnot Lane has devised a treatment for the "kinks" and obstructions that occur in the intestinal canal, by short circuiting the ileum and sigmoid, or even removing the colon altogether; but for general visceroptosis no satisfactory operation has yet been suggested.

It is obviously quite impossible in an address like this to cover the whole field of surgery. Several subjects, in which I have been especially interested, must be left severely alone, great as is the temptation to discuss them. I should like, however, to refer very briefly to just one more modern scientific advance, that perhaps may be said not to belong to surgery at all. I mean treatment by vaccines, and increasing the physiological resistance of the patient. It must be admitted that purely surgical measures have not proved very satisfactory in treating infections. The surgical removal of tubercular organs, for instance, as the testicle, kidney, or lymphatic glands, not only scars and mutilates the patient, but deprives him of the internal secretion of the organs. Further, it does not necessarily cure the infection, which may have originated in, or spread to other parts. The use of vaccines is thoroughly scientific and physiological, and surgeons have eagerly availed themselves of these therapeutic agents. While the results have in some cases been gratifying, in others they have not been all that was hoped. In this, as in all departments of medicine and surgery, we are constantly reminded of our limitations. We are dealing with living organisms, and the mystery of life is still unsolved, and the reasons for the peculiar idiosyncrasies and different resisting powers of individuals remain unexplained. But, decade by decade, even year by year, we see that the circle of our limitations is widened, and we look hopefully to the future, confident that surgery, broad based on physiology and science, will yet win many conquests in the cause of suffering humanity. To enable surgeons to win further triumphs it is essential, as Bloodgood has recently pointed out, that both the profession generally, but especially the laity, should be educated, and kept abreast of surgical progress. Systematic efforts must be made to spread information about the early indications of disease, and the possibilities of their surgical treatment at that stage when it can give the best immediate and permanent results. It may be difficult, even for surgeons well versed in modern methods, and assisted by up-to-date specialists, working in co-operation, to make a positive diagnosis in the very early stages of many cases. To surgeons, however, difficulties and dangers exist only to be overcome, so that, as Hotspur says, "Out of this nettle, danger, we pluck this flower, safety." To the surgeon are peculiarly applicable the words of that poet, who was also a man of science, "The lofty soul can all achieve whose thought is swift, whose grasp is strong."

SURGICAL DISEASES OF THE LARGE INTESTINE—NON-MALIGNANT.

By Fred. D. Bird, M.S.

I have the honour to open the discussion on the surgery of the large intestine, excluding appendicitis and malignant disease.

The alimentary canal appears early in the evolutionary scale and the division occurs into small and large intestine. We have until quite recently looked upon both as beneficent ministers to our absorptional wants, but now the large intestine looms larger every day, and is fast losing its beneficent character in our minds.

A considerable amount of this really awe-inspiring aspect of the great gut is due to our woeful ignorance of the subject.

Seeing it almost daily in the live subject, still owning one of my own, it had been and is a source of much dissatisfaction to me that I know so little about the large bowel, even anatomically.

He would be a bold man who would say what position, or range of positions, can be looked upon as normal for the large intestine to occupy in the abdomen, and as bold would he be who would give a limit in inches for the normal length of this portion of the canal.

In my opinion we must look upon both criteria as varying with different individuals, and in the same individual at different periods.

We have accepted the fact that the urinary bladder gradually descends in relation to the vertical height of the individual, from the fact of its holding water, which is heavy, more or less continuously in an animal which maintains the erect position during a large portion of its existence. Thus we find in old men that the fluid pressure has acted on the portion of the bladder behind the outlet, which is fixed in such a manner as to make this outlet no longer the lowest portion of the bladder.

In like manner, the colon more or less loaded with heavy fæces, must tend to sag after years of the upright position, however good the abdominal muscles may be.

So, looking at the subject from this gravitational point of view, is it any wonder that we find the colon in the lowest parts of the abdomen in many old people, and that the condition of sagging is more seen in women, whose abdominal muscles have their functions largely abrogated, and whose colonic contents tend to be heavier and less constantly moved on than in the male sex.

From what I have observed in operative work on the abdomen, and from watching the progress of bismuth meals with the fluorescent screen in a number of cases, I think the following few statements may be accepted as facts:—

1. The only solidly fixed portion of the colonic gut is the splenic flexure.

I think this must be so, from dissecting room anatomy, from pulling on the transverse colon in the live subject, and finding the resistance to further delivery afforded by the fixation of the splenic flexure, and from the fact that if there is a bismuth shadow in the cæcum or ascending colon and in the sigmoid, there is sure to be a dark shadow high up in the left upper abdomen.

Given this fixity, it follows that any dropping of the transverse colon will cause an increase of angulation of the gut at the splenic flexure, and the more the transverse colon drops the greater will be the obstruction to the free progress of the contents.

Even without ptosis, I believe the great factor in pseudo-ileus to be the natural angulation in this position.

2. The mobility of the cæcum is considerable, and is governed by the size of the cæcum itself and the length of the ascending mesocolon, and its power of having the position altered is conditioned by the attachment of the termination of the ileum. A further complication sometimes exists in the presence of very definite adhesions some 3 or 4 inches from the ileo-cæcal junction in the ileac mesentery. These tie down and angulate the ileum in a way which has a direct influence on the cæcum and its mobility.

Of the reality of this band or wisp of fibrous tissue there can be no doubt. I had noticed it, but until Mr. Lane pointed it out to me last year, I had not appreciated its significance. I think that, like several other bands about this region, it is congenital in origin, becoming more pronounced as life goes on, for reasons difficult to particularise. It is in the track of the descending testicle, the passage of which must give rise to conditions of unrest in the subperitoneal tissue behind the mesentery.

What am I to say about the anchorage of the hepatic flexure?

First we must admit that it often drags its anchor. The surgeon often finds the whole of the colon, from the caput cæci to within a few inches of the splenic flexure, easily to be pulled into his operation wound, and again, the segmented shadows on the screen of the bismuth-bearing colon are often arranged in such a way as to be explained only on the supposition that the hepatic flexure was far away from its supposed anatomical position.

I would like to mention here, that I believe the shadows shown by bismuth on the screen to be a true reflex of the position of the containing bowel or stomach, if care in the placing of the X-ray tube is taken.

Does this dragging concern other organs?

It is often connected with displacement of the kidney, but undoubtedly it may occur when the kidney does not move unduly. I have often pulled on the hepatic flexure and found the kidney uninfluenced by the manœuvre. What, then, is the truth about so-called ligaments connecting the hepatic angle of the colon and the kidney? Undoubtedly, in many cases there is no connection; again, in some persons there is something more than propinquity, and the ptosis of the hepatic angle will tend to displace the kidney. The same philosophy seems to hold good in the cadaver. In many subjects there is no linking band of fibro-fatty tissue; in some subjects such ligaments can be fashioned by dissection.

This being so, it follows that while an operation for fixation of the kidney, which has no regard for the hepatic flexure, may be successful in many cases, in some cases, at all events, success will only follow the anchoring of the hepatic flexure to the posterior abdominal wall.

What effect again has the excursus of the transverse colon on the stomach?

We were pleased some years ago when we found that undue mobility of the kidney caused in some cases transient jaundice from kinking of the common bile duct. This was probably not the whole truth, for it is the dropping of the hepatic flexure and transverse portion of the colon that pulls on the stomach and angulates the pylorus and common bile duct. In some of these cases the kidney will be concerned in the ptosis from actual physical connection.

An operation to remedy this must perforce take cognisance of the colon.

In some cases the stomach is found to have invaded the pelvis with portions of its extent. It is hard to believe that the colon has dragged the stomach down to this distance, but when such extreme ptosis has occurred, the hepatic angle is always, I believe, quite loose.

At first I thought that there must be some fallacy as regards the bismuth shadows in such cases, but subsequent operation has confirmed the reliability of the screen.

Gastroptosis and coloptosis cause, or are associated with redundancy of the stomach and colon. Though I have not yet performed it for ptosis, I feel sure that removal of portion of the colon is the right thing in some cases, mainly because of this redundancy. The irregular coils and obvious surplusage seem to invite removal. This removal can be effected rapidly and without much risk if the splenic flexure and a few inches leading to it are left. In an operation I described in the *Lancet* some years ago, the ileum is divided 4 or 5 inches from the junction, and by opening up the mesentery, the ileocolic vessels can be quickly secured and the colic branches higher up. The key of the position is found in dividing the ileum first and opening the peritoneal leaves. The terminal portion of the ileum, the cæcum, and appendix, the ascending colon, and the transverse as far as to a convenient distance from the splenic flexure, can be removed quickly in this way with the application of but very few ligatures. An anastomosis of the ileum and transverse colon can be made in a manner to suit the operator.

I have performed the operation a number of times for malignant disease with operative success. It leaves but a short uphill traverse for the intestinal contents, very much less than the long up-grade from the pelvis up to the splenic flexure. Some uphill in the stream is inseparable from our upright position. Personally, I think that removal of the whole colon can but seldom be indicated. The danger increases much with the ablation of the splenic flexure, because of technical difficulties, but I do believe that the modified operation I recommended has a real métier in the treatment of gastro-coloptosis.

Without being able, from want of personal experience, to subscribe to all Mr. Lane's contentions with regard to the peccability of the large bowel, I certainly saw, at Guy's Hospital last year, a number of cases in whom various ailments seemed to be much lessened by colectomy and anastomosis.

3. The third fact in connection with the colon is that its position in the abdomen is much influenced by gravity. This is beautifully demonstrated by the X-ray screen. A colon with bismuth-laden food in it may show by its shadows as a pelvic organ in the upright position; put the examinee on a horizontal table with the tube beneath, and a totally different picture is visible, the colonic shadows now being seen above

the umbilical level. Tilt the foot end of the couch, and a further displacement occurs. This mobility, of course in less degree, obtains in normal individuals, and a recognition of this fact is often of great technical service in removing the appendix. The colon may be superimposed upon the cæcum, and the surgeon may be able to get at the appendix through a small opening only by having gravity to assist him in displacing the colon.

Again, much relief is given to some cases of enteroptosis, by the application of cross strips of Mead's plaster, but these must be applied after the colon has been induced by gravity to occupy a high position in the abdomen.

The V-shaped transverse colon is commonly seen in post mortems and operations, but recently at the Melbourne Hospital, the examination of a dead body revealed a W-shaped transverse colon, which I believe is a great rarity.

The congenital factor must not be lost sight of in any consideration of the causes of enteroptosis. In young subjects, both small and large intestines will deliver through a median incision very much more easily in some cases than in others.

In considering the ætiology of abdominal ptosis, many discrepancies and apparent contradictions occur. Thus, want of intra-abdominal pressure due to poor development of the abdominal muscles, must, without doubt, be counted as, at all events, a predisposing factor in most cases of ptosis, yet we also, without doubt, must admit that some persons, although gifted with good abdominal muscles, are handicapped by ptosis.

Again, we see constipation without ptosis, and ptosis without constipation, although we can but admit that in most cases there is an important interacting association between the two conditions. Other disharmonies could be mentioned, and all point, in my opinion, to some as yet unrecognised factor in the causation of abdominal ptosis.

Many operations other than those mentioned have been devised to alleviate the symptoms caused by the dropping of abdominal organs; all of them have done some good in some cases; even so simple a procedure as stitching the great omentum high up on the anterior abdominal has done much good in one case I know.

The common denominator in these cases must be the neurotic factor, which is present in various strengths in every case.

The part played by the muscles is, in my opinion, underestimated, and we ought to encourage an increase in their tone and efficiency. The diaphragm and the levator ani must contribute to the production of a plus pressure inside the abdomen, as much as the recti and lateral muscles. It is too much to hope that muscular action can cure ptosis, but in most cases it can prevent its formation and certainly its increase.

The next, to me, most interesting condition connected with the colon is seen in those cases where a large granulomatous tumour mass is formed in response to the presence of some micro-organism, that of tubercle or of syphilis, or the ordinary pyogenic cocci with bacillus of the colon.

These formations are very puzzling in diagnosis, as well after as before operation. An endeavour should be made, by every means of diagnosis, to arrive at a diagnosis, for while they all simulate malignant disease, only one of them, and that the tubercular form, requires a large operation.

The granumata due to ordinary cocci are usually found in connection with a foreign body which has perforated the gut, and introduced germs into the subperitoneal tissue. The most likely place for this to occur is in the descending colon, and the commencement of the sigmoid loop. After a time abscess may form, and as abscess is not uncommon in connection with carcinoma in this region, a further diagnostic difficulty ensues. In some cases of perisigmoid inflammation, abscess and not granuloma forms first, the condition being analogous with perityphlitis on the opposite side.

All such cases should be approached operatively as if the inflammation were in the subperitoneal space. The abscess can generally be opened from the retroperitoneum.

That the sigmoid colon is an area of unrest may be gathered from the very frequent presence of adhesions in the vicinity, and its adhesive association with the broad ligament in inflammatory conditions within that fold is always important, as the junction of the bowel and the broad ligament is often physically stronger than the free bowel.

Syphilis, however, plays another rôle in the sigmoid, where syphilitic ulcers may give rise to minute perforations with subsequent abscesses which burrow and burst, giving rise to sinuses, whose origin it is very hard to determine. The difficulty of differentiating these sinuses from those due to retroperitoneal abscess is very considerable, but the syphilitic ones are likely to be further away from the iliac crest than those of retroperitoneal origin.

Volvulus of the sigmoid flexure has interested me much. I believe it be of commoner occurrence than is usually thought. Like the big volvulus, it generally occurs in middle-aged men of constipated habit. The symptoms in the cases I have seen, have not been urgent, but the obstruction has been complete, neither fæces nor flatus passing. The mildness of the symptoms is due to the twist not causing much interference for a long time with the blood supply. This is one of the forms of intestinal obstruction which sometimes rights itself, and the surgeon may untwist the gut in delivering it through the incision and then be at a loss to account for the symptoms. I always watch very carefully for this when operating in the sigmoid region for obstruction. This volvulus tends to recur, and there seemed to me to be in each case a redundacy of the sigmoid portion.

Myxœdema produces very confusing tumours in the omental and colonic areas in some cases. The myxomatous masses are as a rule multiple, and the diagnosis will be almost impossible unless the general condition, which may be poorly marked, is recognised. The danger is that a very large operation may be performed, or a hopeless prognosis given.

Cases of multiple adenomata and multiple diverticula of the large gut have come my way, and the latter sometimes give rise to abscess and sinus.

In connection with hydatid cysts, several points of interest occur to me. So frequently did it arise in the experience of Davaine, writing in Paris some seventy years ago, that he thought the bursting of a suppurating hepatic hydatid cyst into the transverse colon, with subsequent spontaneous cure, to be the usual ending of such a case.

Hydatids are often found in the closest proximity to the large gut, and in removing omental cysts the surgeon has to exercise much care, with the application of his ligatures, not to pucker up the wall of the large bowel, but it is only rarely that the echinococcus is found actually in the wall of the colon. Two such cases have I seen, and Dr. Honman, of Melbourne, has recently reported another.

A point I wish to bring forward in relation to hydatid abutting on the colon, is the occurrence of intestinal obstruction due to its presence. It has been a source of wonder to surgeons that the maze of omental and peritoneal cysts, so often seen, does not produce obstruction. One form, however, as far as I can make out, does interfere to the point of obstruction, with the colon, and that is when a cyst develops in the mesocolon and goes on growing. In such a case, intestinal obstruction is likely to occur. In some cases a cyst growing in the retroperitoneum opens up the mesocolon in its advance, and will produce the same effect.

I have purposely not touched upon the subject of intussusception, as it would amount almost to impiety to do so in the city which owns Mr. Clubbe.

THE SURGICAL TREATMENT OF MALIGNANT DISEASE OF THE LARGE INTESTINE.

By H. Critchley Hinder, M.B., C.M. (Syd.).

In order to impress upon you the very great importance of this subject, I shall quote you a few statistics, illustrating the great frequency with which malignant disease of the large intestine is met.

Of the total number of general autopsies as collected by Zemann, Maydl, Muller, and Nothnagel, amounting in the aggregate to 69,083, there were 5,796 deaths from carcinoma, about 8·4 per cent. Of these 5,796 deaths from carcinoma, 1,296 occurred in the intestinal tract.

So that you will observe that 22·35 per cent. of all carcinomata occur in the intestinal tract. Of these again, 2·7 were in the cæcum, 10·11 in different parts of the colon, 6·405 in the sigmoid flexure, 20·22 in the rectum.

With or without accurate statistical data I feel sure that most of you who see a large number of patients will bear me out when I say that malignant disease of the large intestine is so common, particularly among patients getting on in years, that we have need to constantly bear this fact in mind when investigating the causes of abdominal symptoms.

The majority of the malignant growths in the large intestine are cancers. Sarcomata are extremely rare. Cancers growing from the region of the anus are usually epithelial in type, with a tendency to spread towards the perineum and infect the inguinal lymphatic glands. Those originating higher up the bowel are usually cylindrical celled or adeno carcinomata. The point of great practical importance to us as surgeons is that these cylindrical celled growths are not very prone to take on early metastasis. Iverson, who investigated forty-seven rectal carcinomata *post mortem* found the glandular organs free in twenty-one of them.

Most other pathologists are in agreement with this opinion.

Personally I have always held to the view that malignant disease of the large intestine is less likely to recur than is malignant disease in other parts, so that in anything like early cases the prognosis is very favourable. This opinion is based on clinical results.

My own experience has induced me to take a very favourable view of all early cases. Some of those with a history of several months who submitted to operation are alive now after an interval of from nine to twelve years. Needless to say the fact that we may look upon the disease with a favourable eye should not deter us from using our utmost endeavour to urge surgical treatment at a very early date, for, clinically, no one can say what may be the degree of malignancy of the growth under consideration.

I have myself remarked, and it has been noted by others, how very rarely the patient suffering from malignant disease of the bowel presents himself until the disease is well advanced.

Hemmeter states that in sixty-six cases of rectal cancer, forty-eight did not consult a physician until four months had elapsed, and doubtless they had been afflicted some time before actual symptoms became manifest. When we come to consider carefully the history of each individual who presents himself of us, the conviction is forced upon us that in all probability there is in many of them a precancerous stage which needs our most careful study, and which will present to us the most hopeful solution of the difficulty we experience in diminishing the mortality from this dread disease. It is true, as I have already intimated, the fault lies to a large extent with the patient himself, but without doubt, education in matters relating to personal hygiene will induce a better appreciation of the danger of neglecting any departure from the normal. Although the history of some patients may be short and without incident, there are others in which the patient can give a distinct history of chronic colitis or occasional attacks of obstruction with pain going on for from two to seven years. It behoves us, then, in this and all other cases of chronic irritation, to adopt rigorous measures to reduce the tissues to a normal condition as early as possible, or if necessary, to open the abdomen and discover the precise nature of the trouble.

It is unfortunately true that many a patient does not reach the surgeon until he is suffering from intestinal obstruction. This, too, I am glad to say is often not the fault of his medical attendant, but the fault of the patient himself, who imagines that he is experiencing an attack of flatulence and only needs a little aperient to put him right. Our treatment, therefore, frequently resolves itself into dealing with a case of intestinal obstruction as well as the removal of a malignant growth. If I might venture to offer the suggestion, it would be as well if we were to hinge our discussion on certain methods of procedure.

Should we relieve the obstruction and remove the growth at the same time, or should we merely relieve the obstruction and attack the growth at a later period?

In either event, what would be the best method to adopt?

It will greatly simplify matters if I simply give you my own reasons and my own methods. We must also take it for granted that from the age and the history of the patient we have a strong impression that he is suffering from malignant disease of the bowel.

Let us suppose that the patient is found to be suffering from acute intestinal obstruction. We will in very many instances be assisted by the patient's own statement as to the probable situation in which the obstruction is to be found.

If the patient is very greatly distended and in dire distress, all we should aim at is the relief of the imminent danger which threatens him. Let a 1½ inch or 2 inch incision be made through the skin just above and internal to the ant. sup. spine of the ilium on the right side, split the muscle with the handle of the knife, and open the peritoneal cavity. First see if the cæcum is very distended. If this is the case it is fair to assume that the seat of obstruction is on its distal side. Such being the case, proceed to establish a fæcal fistula in order to drain the bowel. Draw out a nipple of the cæcum and stitch a portion the size of a shilling to the parietal peritoneum with a fine catgut running suture. Keep hold of the centre of this piece of bowel with the forceps, and before making an opening with a tiny sharp-pointed pair of scissors, pack a narrow strip of gauze round the line of suture in order to save any leakage past the suture into the peritoneal cavity. A tube is inserted a little larger than a No. 12 catheter, and the cæcal contents will drain freely away, or drainage may be assisted by running in a little warm water now and again in order to clear the tube and dilute the fæces.

The patient who has been worried by pain and whose resistance has been lowered by the absorption of toxins has now time to recover. His distended, engorged, and unhealthy intestine recuperates, so that after a few days both he and his bowel are in a fairly fit condition to stand severe operative measures. A median incision is made in order to locate precisely the situation of the growth and the presence or absence of metastases. The abdomen is closed and, if necessary, a fresh incision is made right down on to the growth with a view to its removal. Should removal be inadvisable, it is astonishing how well patients may get on with this small cæcal opening. By means of the tube the bowel may be washed out night and morning, the tube being retained during the day, doubled over and occluded, and held in place by a dressing. Authorities repeatedly mention the method of removing the intestinal contents in a case of intestinal obstruction by making an incision into the bowel and milking or draining segment after segment. I tried it once, and it appeared to me to be a very unsatisfactory procedure. The patient is usually so low that the time taken must necessarily be long, and the shock attending the manipulation is ill borne by these patients. The contents of the bowel are so virulently infective, and the chances of peritonitic infection so very great, that if in addition to all this a resection of the bowel were to be considered, I for one would be rather anxious about the final issue. A person suffering from acute intestinal obstruction is always a very poor subject for extensive operation.

Some recommend colotomy as a preliminary measure, but a cæcal fæcal fistula made, not through an aponeurotic structure, but through muscle in the manner suggested, will almost invariably close with very little trouble, and is in every respect preferable.

It would be absurd for me in the short time at my disposal to go into the details of the very large number of methods in vogue for the removal of malignant growths of the large intestine, or for dealing in a palliative way with such growths. I must needs content you with my own opinion.

I have already mentioned the formation of the fæcal fistula and how well and comfortably it acts. Should the patient with an inoperable growth find such a fistula irksome, it is an easy matter in most cases to open the abdomen, and with a short circuit get past the obstruction. A patient of mine at the present moment has a fistulous opening in the cæcum. He is 76 years of age, with a failing heart. I believed him to be suffering from an obstruction due to malignant disease, but since the fistula has been established his bowels frequently act per anum, but he keeps the fistula open as a safeguard. I have seen similar cases which lead one to believe that the acute obstruction is very like the induced swelling which brings about an acute retention in stricture and hypertrophy of the prostate. An obstruction is present, but a little inflammatory œdema is required to bring about a complete closure; so that after a relief has been established by means of a fistula, the bowels begin to act again per anum.

Though I am strongly in favour of the establishment of a preliminary fistula for the relief of the acute obstruction, there are times, especially if the cæcum is not distended, when it may be deemed worth while to open the abdomen in order to make sure what is the precise cause of the obstruction, and this should certainly be done if the distention is not too great, and if there is an element of doubt as to the cause of the trouble. I may state that I have operated on three cases of intestinal obstruction due to gall-stones in elderly people, and in each case the abdomen was first opened in order to locate the site of the obstruction, partly because the patient's condition did not contra-indicate such an examination, and partly because of the slight degree of cæcal distention.

Let us next proceed to consider the removal of the growth, and in doing so first deal with the operative procedure suitable for the removal of a growth in any part of the bowel from the cæcum to the sigmoid.

The incision should never be larger than what is absolutely necessary to get well clear of the growth. First take the tumour in hand, stretch its peritoneal attachment taut and incise the peritoneum on the parietal side, well clear of the growth, then with a dry swab press back the mass with its leash of vessels towards the mid line. Incise the peritoneum similarly on the visceral side, and it is astonishing how a tied-down growth will become freed and further manipulation become facilitated. Proceed to tie off the blood supply, getting well back so as to be free of the glands likely to be involved, double clamp the bowel above and below, well free of the tumour, and cut away between the clamps.

Undoubtedly the best way to reunite the bowel is to tie off the vessels on each stump, turn in the ends, and with a purse string or a row of continuous suture close it completely. Then lay the upper and the lower portions alongside one another and make a lateral anastomosis. Unfortunately, however, this is not always possible. My objection to the end to end anastomosis is that there is far too great a danger of leakage, where the bowel remains uncovered by peritoneum. This situation is difficult to get at, and sometimes do what you will the leakage will take place there.

In order to give a good anastomosis some recommend that when the growth to be removed is in the cæcum or in the ascending colon, that the whole of the cæcum should be removed in order that the ileum should be free to be drawn up and anastomosed laterally with the hepatic flexure. The result so far as the bowel goes is good, but it would, perhaps, mean the making of a very large rent in the abdominal wall, and possibly may be a more severe tax upon the patient, and I have certainly seen excellent results from a mere closure of the ends and an anastomosis of the ileum with the distal end of the ascending colon.

The transverse colon gives us a good opportunity for lateral anastomosis on account of its length, but I have had cases where the removal was so free that it was necessary to connect the ileum with the sigmoid and the result was good.

I have several times removed a portion of the descending colon, followed by an end to end anastomosis, and almost invariably the operation was followed by a leakage of fæces. After the suture was completed the omentum was brought down and lapped round the gut so as to shut it off from the general peritoneal cavity. In addition to this a layer of gauze was passed down, and a thick soft tube, so that when a leakage did occur there was ample provision for any accident which might take place. I am sorry to say that this method was not adopted until I had lost a case owing to the posterior part of the anastomosis giving way. This was the only case I ever lost.

The method which has been described, of drawing out the loop of bowel together with the growth, cutting away the growth situated at the end of the loop, and fastening the upper and lower sections of the bowel laterally so that they were like a double-barrelled gun with the muzzles projecting from the wound, is fairly safe, but the final closure is not without an element of risk, and at all events the method is crude, and it should, and of course can, be improved upon.

In this region, and also in the sigmoid and upper rectum, I have adopted a method which I shall now describe to you. It is original, as far as I am concerned, but it may have been described before for all I know. That, after all, is a matter of little importance; but I feel confident that it is not generally known, and what is more, the method appears to me to be reasonable, simple, and efficacious.

Suppose the growth is in the lower sigmoid, an incision is made which will control the field of operation, but if the Trendelenberg position will in any way help, by all means adopt it. In fact, such a position will be applicable for all sigmoid cases, but is not of much value in dealing with the descending colon.

Let us suppose that the growth has been isolated, the peritoneum incised, and the vessels tied, and glands if necessary removed so that we have simply the cut ends of the bowel to deal with. The area or field of operation is packed round with gauze pads so that the subsequent manipulation will not bring about any peritoneal contamination. The clamp is removed from the rectal end of the bowel. The bowel is held in position by two artery forceps. A very long pair of forceps is passed down the rectum, emerging at the anus. With this forceps grasp a piece of soft, fairly pure rubber tubing, 1 inch internal diameter, and having an obliquely-cut end. Draw the tubing up until about 3 inches project from the upper end of the rectum. The clamp is now removed from the cut end of the sigmoid, it is grasped by two pairs of forceps, and the 3 inches of rubber tube is thrust into it, or the sigmoid is drawn down over the tube, which amounts to the same thing, so that the full 3 inches of tubing are within the sigmoid. A piece of catgut is tied tightly round the lower end of the sigmoid so as to grip it firmly on to the rubber tube. It should serve to stop any oozing which may take place from the cut end, and will hold the sigmoid fast in place. This I have found to be hardly enough; in fact, I am sure that it is much better to place in addition at least four sutures through the lower part of the sigmoid and the tube, so that this lower end should be firmly attached to the tube. Next take the rectal end and draw it up so as to cover the sigmoid for at least an inch, turn in its raw edge; if there is any bleeding tie a piece of catgut round the gut close to its margin, gripping the tube and gut beneath, still further roll up the rectum so as to double over this cut rectal end, and run a continuous suture of catgut round so as to get a good approximation of the peritoneal surfaces. The object is to give a good broad approximating surface. The large tube helps union by conducting away fæcal material and gas, preventing at the same time any strain on the newly adjusted parts by the pressure on them, and consequent distention, which would necessarily follow under ordinary circumstances. The rubber tube should, as an additional precaution, be made fast by running a safety pin through it outside the anus, and tying it in with two threads or tapes attached to the pin and passing in front and behind the thigh to be attached to a waist-band. The first patient I operated upon after this method was a fairly stout man, who had a malignant growth almost as large as my fist in the upper part of the rectum. It could just be brought in contact with the manipulating finger in the rectum by pressing hard down on the abdomen. As a precaution I made two stab wounds through the abdominal wall and passed two drainage tubes, one on each side of the united gut. They were soon removed, as there was no need for them. Some fæces escaped by the side of the tube at about the second day. The tube itself came away about the sixth day, and the patient experienced no trouble, but was remarkably comfortable the whole time. The catgut sutures holding the tube to the sigmoid rot away very quickly, so that there is no difficulty in getting rid of the tube.

The second case in which I used this method was in a patient, a man about 50 years of age, with acute intestinal obstruction. A cæcal fistula was made, and at the end of a week the removal of the growth was effected. It was close to the splenic flexure, and was consequently rather difficult to get at. The same large tube was used in the manner I have described, but only one suture was used to attach the bowel to the tube, so that at the end of thirty hours the tube came away, and there has been some fæcal escape. The tube was hardly suitable owing to the nearness of the splenic angle. This method can, at all events, be strongly recommended for any case involving the rectum, the sigmoid, or the descending colon. I first devised this scheme about fifteen months ago, but it was only four months ago that I came across a suitable case. This patient is in good health in every way.

I must confess that any method of attacking the growth through the sacrum has not appealed to me. I have operated in this way, but always with the feeling that unless the growth were very close indeed to the anus the abdominal route would have given me a much better opportunity of getting well clear of the growth. I have also removed a growth by the combined abdominal and perineal method, drawing the sigmoid end out at the flank and making an artificial anus, but I feel sure that with the aid of the tube the sigmoid could be drawn down, if even there were only an inch or an inch and a half of the lower end of the rectum left.

In dealing with malignant growths it is very necessary that the whole field likely to be involved should be under easy observation, and the abdominal route alone satisfies this condition. The reason why we hear of a certain number of cases doing well after operation by the sacral method is most likely because the growth so often remains a purely local affair for a considerable period.

I have purposely made this paper a short one because I did not consider it worth while at such a meeting as this to deal with signs and symptoms which are so well known to you all, but I have endeavoured to pick out a few important and debatable points worthy of your consideration.

No doubt in malignant disease of the large intestine we have a fine field for operative treatment which promises to give us a good percentage of cures owing to the peculiarly mild degree of malignancy in a large number of cases. Let us, therefore, so urge upon general practitioners and physicians the great possibilities of radical treatment that they may be the more keen in doubtful cases to give us an opportunity of discovering the trouble in its very early stage. Many men speak of exploratory surgery as a confession of ignorance. I think we admit our ignorance, but I am quite confident that much more harm has been done by waiting than by operating, and with the diagnostic means at present at our disposal it would be well if exploratory surgery were practised much more frequently than is the case at present. I have never regretted opening an abdomen, but I have often regretted that I had not had an opportunity of doing so at an earlier date.

ON THE SURGICAL PATHOLOGY OF THE LARGE INTESTINE WITH SPECIAL REFERENCE TO CARCINOMA.

(A contribution to the opening of a discussion on the Surgical Diseases of the Large Intestine at the Ninth Australasian Medical Congress, Sydney, September, 1911.)

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The growing interdependence of pathology and surgery is a fact of good omen for both. Time was when pathology began and ended in the *post mortem* room. But modern pathology is concerned chiefly with *ante mortem* processes. The study of disease more often begins, and hence more often ends, with the living patient. A cause and a result of this development is that the practice of surgery, like the practice of medicine, has become more scientific. The successful surgeon of to-day must be not only a good anatomist and physiologist, but above all a pathologist of no mean order.

In such a brief review, as this must be, of the surgical pathology of the large intestine, I can refer only to certain conditions that have come within my experience, and I take, as the most important, the malignant new-growths.

Incidence of cancer of the intestine in relation to irritation.

In the unknown causation of cancer there is no factor more obvious, no factor more constant, than irritation (Refs. 1 and 2); and the influence of irritation in determining the onset of cancer is nowhere better illustrated than in the intestine. For the sites at which cancer of the intestine is most frequent are just the positions at which irritation of the mucosa is greatest. Now, irritation of the intestinal epithelium is conditioned partly by the form and fixity of the channel, and partly by the physical state of its contents. The relative immunity from cancer of the small intestine between the duodenum and the ileo-cæcal valve is probably due to the fact that irritation is reduced to a minimum by the easy passage of its semifluid contents along a muscular tube of uniform calibre suspended so as to avoid fixed turns. But irritation is induced by the configuration and by the fixation of the tube at the ileo-cæcal valve and at the flexures of the large intestine, and is still more aggravated by the increasing consistence and accumulation of the contents in its lower part. And in the large intestine cancer is common, and is located most often in the sigmoid and rectum, where irritation is at a maximum, and next at the cæcum and flexures.

The appendix, however, is seldom the seat of cancer, and yet inflammation and irritation are very common there. This is difficult to reconcile with the known close relation between cancer and irritation, except on the hypothesis that irritative conditions of the appendix do not last long without causing symptoms of urgency, so that death, or the surgeon, intervenes before a cancer can develop.

It is interesting to note that women are somewhat more liable to carcinoma coli than are men (Ref. 3); and that women, more often than men, suffer from habitual constipation and the resultant greater irritation of the lower bowel.

The age incidence may be misleading as an indication of cancer of the large intestine. For in this region cancer is not an uncommon cause of obstruction at any age, even in young adults. Probably there is no other delicate epithelium in the human body exposed to such unremitting irritation as the lining of the large intestine, and this may induce a premature senescence of tissue and an earlier genesis of cancer in a certain proportion of cases.

General characters of cancer of the large intestine.

A. *Histological.*—Sarcoma is rare. True carcinoma is relatively common. The predominant form is an adeno-carcinoma, originating from the epithelium of Lieberkühn's crypts. The adenomatous arrangement is usually of the inverting type, and in most cases there is associated a greater or less degree of "colloid" change in the carcinomatous epithelium. The density and relative amount of the stroma varies in different growths, and in different parts of the same growth.

B. *Anatomical.*—The commoner anatomical forms may be grouped as (*a*) annular, (*b*) tubular, (*c*) fungating, and (*d*) eccentric, in that order of frequency. In the *annular* forms a narrow ring of dense cancerous new-growth invests and constricts the bowel, causing progressive chronic obstruction which may at any time become acute. In the *tubular* forms a more or less rigid tube of new growth extends several inches along the bowel. The actual narrowing is usually less than in the former type, but the mere rigidity of the tube is contributory to the obstruction. In the *fungating* form softer masses of more rapid growth project and may ulcerate into the lumen, and may or may not give rise to noticeable obstruction. In the *eccentric* form an active proliferation of cancerous tissue is localised in the wall of the bowel, and may come to form a palpable tumour with or without obstruction.

C. *Clinical.*—The two pathological results that most commonly give a clinical sign are obstruction and tumour. Some degree of obstruction is present in nearly every case, and evidence should be most carefully sought in regard to it, since it is by far the most important indication. Palpation is of less value in diagnosis except in so far as it may reveal the fæcal accumulation above an obstruction, or unless the growth is within reach of the finger in the rectum. Otherwise a palpable tumour is rarely met apart from advanced conditions.

Mode of spread of carcinoma of the large intestine.

The spread of cancer in the alimentary tract reveals two distinct processes, one of which is, not to my knowledge, recognised. This is the local extension of the cancer by the progressive involvement of adjacent cells in the cancer process. Side by side with the cancerous new-growth, there can be seen cells of the mucous membrane becoming cancerous *in situ*. Epithelial cells, in process of transition to cancer cell, are seen to become more rounded, the nucleus more central, the cytoplasm more granular; but the most noticeable change of all is that the cells acquire an increased affinity for cytoplasmic stains. The appearances are very striking, and are such as to suggest that some malign influence passes by direct contagion from cells already cancerous to healthy cells, gradually transforming them also to cancerous types. Several instances of sarcomatous development in the stroma of experimentally propagated carcinoma might be interpreted in the same sense. It may, however, be that the unknown cause of the cancer process continues to act, and to implicate adjacent cells, long after the earliest cancer cells have been formed. In any case the local spread of the cancer, *so far as the mucosa is concerned*, would appear to be due to a process of direct extension to contiguous cells, rather than to an undermining of the mucosa by an invasion of cancer cells from a focal growth. And, so far as I have been able to see, this holds good for the initial transverse extension in the mucous membrane as well as for the extension in the long axis of the gut.

The other mode of spread is well recognised, and may be described as the *dissemination of cancer cells* as distinct from the local *extension of the cancer process* indicated above. This dissemination is in the first instance local, but tends to become generalised. It is best realised by thinking of a cancerous invasion as analogous to a chronic bacterial infection (*e.g.*, tuberculosis), the infective agent being the cancer cell in place of the bacterium (Ref. 2). It is by this process that the cancer cells invade the deeper layers of the mucosa, penetrate the muscularis, and reach the lymphatics of the subserosa. It is by this process that the cancer cells, following the lymphatic spaces and channels of the intestinal wall, form most commonly an annular investment of the bowel, and come to invade the lymph glands into which the affected segment drains. In all these respects the dissemination of the cancer cells corresponds with the spread of tubercle bacilli in the development of tuberculous ulceration of the intestine. The cancer cell travels by the same spaces and channels as the bacillus, it is deposited and multiplies in the same positions.

The general lymphatic dissemination is most significant for the surgeon. It has recently been shown by Jamieson and Dobson (Ref. 4) that the lymphatic drainage of the large intestine follows a certain orderly plan, and by Clogg (Ref. 5) that the dissemination of carcinoma coli proceeds on the same anatomical lines. These observations, of great practical importance as well as of scientific interest, have been summarised by Makins (Ref. 6) from whom this account is taken. According to Jamieson and Dobson, the entire lymphatic basin of the large intestine is made up of a number of more or less definite "lymphatic areas," and in each lymphatic area there are more or less definite systems of glands through which the lymph stream passes. The main "lymphatic areas" are indicated by the main arterial branches, since the lymphatics of any segment of the colon correspond in their distribution with the arteries supplying that segment. Hence four lymphatic areas may be recognised:—(1) *ileo-colic*, (2) *middle colic*, (3) *left colic*, and (4) *inferior mesenteric*. The right colic artery, being inconstant in origin and having few glands along its course, is not assigned an individual area.

The lymph glands connected with each area are divided into four distinct but intercommunicating groups:—(1) *epicolic* glands, contained in the appendices epiploicæ, and lying on the wall of the bowel; (2) *paracolic* glands, arranged parallel to the mesenteric attachment of the bowel, between the intestine and the arterial arcades and on the arcades; (3) *intermediate* glands, distributed along the trunks of the arteries about midway between their origin and the gut; and (4) *main* glands, grouped around the main trunk of the artery near its origin. The intermediate glands for the most part receive lymphatic vessels from the epicolic and paracolic groups, but some lymphatics may pass to them directly from the bowel itself. The "main" glands collect lymph chiefly from the three peripheral groups, though in certain regions, notably in the lower part of the inferior mesenteric system, they drain directly from the intestine. The main glands discharge centrally into the lumbar and mesenteric glands, and have lateral connections with other systems, such as the splenic.

The great value of these observations lies in the fact that they indicate the procedure by which complete removal of colic carcinoma is most likely to succeed—by extirpation of the whole lymphatic area involved. They also serve to define the limits within which complete removal is possible, since implication of the main glands makes it probable that the cancerous invasion has extended beyond reach. Moreover, they help to explain how it is that in cancer of the intestine there is a longer period, than is usual in cancers elsewhere, between the probable onset of the growth and the stage at which complete removal first becomes impossible. This relatively long period of grace would appear to be due partly to the sequence of lymphatic barriers delaying the advance of the cancer cells, notwithstanding occasional short-circuiting, and partly to the moderate degree of malignancy inherent in this type of new-growth, the cells of which are characterised by a capacity for local penetration rather than for general dispersion. It is important, however, to note that intestinal carcinoma may grow for a long time without being recognised, the signs and symptoms being masked, so that between its recognition and the time limit for successful removal there may be no interval remaining.

Indications of Treatment.

Two great advances in the treatment of carcinoma were made by scientific surgery when it was recognised that removal of the growth should be done earlier, and done more thoroughly. Earlier removal was made possible by more careful diagnosis; more use was made of the microscope in doubtful conditions; less trust was put in naked-eye appearances and in expectant methods. More thorough removal became possible when the surgeon acted on the knowledge that every cancer cell he left behind might be the starting point of further growth; when he learned not to implant cancer cells in the tissues at operation; when he studied the lymphatic drainage of the structures involved in the growth, and tried to excise the whole lymphatic area. In the large intestine peculiar difficulties attend the early recognition of carcinoma, and, as we have seen, it is only within recent years that the anatomical and pathological data for complete removal have been forthcoming.

All work goes to show that the only chance of cure is extirpation—radical and complete. It is true that we are not in a position to say that complete removal is a certain cure of cancer, for it is conceivable that the unknown causes of cancer may continue operative and may generate a fresh growth near the site from which the original growth was taken. Nevertheless, in our present state of ignorance, it is only right that we should look upon every recurrence of an identical growth as evidence of an incomplete removal rather than a fresh recrudescence. Now, complete removal of carcinoma, whether in the colon or otherwise, makes two demands of the surgeon:—(1) an early recognition of the need for operation; (2) an operative technique based on a scientific knowledge of the life-history of the cancer cell.

I take it that every surgeon is fully seized of the fact that the best chance of recovery lies in early operation. But early operation depends on the early recognition of what?—of the presence of new-growth? I should prefer to say that an operation is indicated as soon as the surgeon is satisfied of the probability of new-growth. For in the large intestine there can be no certainty of carcinoma within the period during which complete removal is possible. There is no sign nor symptom diagnostic of cancer, and from this position a portion of suspected growth cannot be submitted to a pathologist. The diagnosis rests on the calculation of a probability, and the first condition of successful surgery is the acumen with which this calculation is made. If the surgeon waits until the probability increases to a certainty, he no less surely waits until the chances of successful operation diminish to nil. What I said of cancer at our Congress six years ago is no less true to-day. If there be any doubt, give the patient the benefit of the doubt—and operate. In these days of modern surgery an exploratory operation is little more than a trifling and temporary inconvenience.

But however alert the surgeon may be to the fatal consequences of delay, he will from time to time be confronted with more or less advanced cases. It may be that a physician has toyed with the case for some time. More often the patient has neglected to seek help until late in the disease, and there is nothing more tragic than the futility of such endurance. Then complete removal may no longer be possible, or may be possible only by an extensive operation scientifically designed to eradicate all the cells of the growth. Even when it is obvious that removal of a carcinoma cannot be complete, it should be made as complete as possible. For cancer cells do not have an unlimited duration of life. In a few instances they have been known to die out before they have caused the death of their host. Unfortunately, this happens only exceptionally; but in dealing with cancer, the surgeon should give the patient every chance, even the most exceptional. Hence in an advanced case he should not be satisfied with a perfunctory operation for the relief of obstruction, but should do the most radical extirpation that he, and the patient, can stand.

Conditions simulating carcinoma of the colon.

Of the conditions that may simulate carcinoma coli there are two most noteworthy:—(1) tuberculosis, and (2) chronic inflammation. The particular form taken by a tuberculous infection depends on two main factors—the number and virulence of the bacilli on the one hand, and the resistance offered by the affected tissue on the other. The interaction between them probably determines whether the lesion will take the form of a proliferation of tissue terminating in necrosis (*e.g.*, hyperplastic tuberculosis), or the form of a more chronic induration leading to contraction of tissue (*e.g.*, cicatricial stenosis of the bowel). The anatomical site of the process also conditions the form of the tuberculous lesion. Thus infection from the lumen of the bowel may be concentrated in the sub-mucosa, causing ulceration of the mucous membrane; or the infecting bacilli may pass more deeply into the intestinal wall and lodge in the subserosa where proliferative changes lead to hyperplasia. But whether the lesion be mainly cicatricial or mainly hyperplastic, the clinical resemblance to carcinoma is so close as to make the differential diagnosis largely conjectural. This matters little, however, since operative interference is indicated in any case; but when the lesion is exposed to view, it becomes important to decide. If it should prove carcinomatous, then the corresponding "lymphatic area" should be excised; but if it should be tuberculous, a limited excision may be more safely practised. For abdominal tuberculosis differs from pulmonary forms in that it appears to benefit by being stirred up, so that improvement may continue although other tuberculous foci are left behind. The different tissues (abdominal and pulmonary) appear to have different capacities for local resistance, and there may also be differences in the virulence of the infecting bacilli, *e.g.*, bovine types in abdominal lesions and human types in pulmonary.

In regard to other chronic inflammatory conditions I have had no personal experience, and I am indebted to Makins (Ref. 7) for the following account. A few are possibly tuberculous, but most belong to the category of "diverticulitis" of American surgeons. The association of false diverticula of the intestine with obstruction of its lumen was first described by Sydney Jones in 1858, and in more recent years there have been recorded operations on cases in which the signs and symptoms produced by inflammatory conditions have simulated in an extraordinary degree those of malignant disease. Again, the mysterious disappearance, after simple exploration, of tumours deemed to be malignant and inoperable by surgeons of experience, and the surprising results after colostomy in similar circumstances, all tend to show that inflammatory conditions of the large bowel, giving rise to obstruction, are by no means uncommon (*cf.* Moynihan). In five cases of peridiverticulitis operated upon by W. J. Mayo, the disease was found to be limited to from 4 to 8 inches of the gut, and had led to chronic inflammatory thickening, chiefly in the muscularis and subserosa. I would suggest that some of these conditions may really have been carcinomata of an atrophic type, such as are not uncommon in the pyloric region of the stomach. In the older parts of such tumours the epithelial elements may completely disappear, and only chronic inflammatory tissue remain ; so that, unless a thorough microscopic examination is made, involving the outlying parts of the induration its carcinomatous nature may not be revealed.

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THE TREATMENT OF SURGICAL SHOCK.

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Before opening the discussion on the treatment of Surgical Shock, I wish to thank the Surgical Committee for the compliment paid to me, and to express a hope that my few remarks may merit their confidence. The Committee is to be congratulated on its choice of this subject for discussion, for, fortunately, the prevention and treatment of this great emergency in surgical work is now being put on such sound lines, that it is already beginning to lose half its terrors.

The two great dangers of all surgical operations are sepsis and shock. We have almost eliminated the danger of sepsis by sound preventive measures. When this is said, we are but paying a just tribute to the great name of Lister, certainly the greatest Englishman alive to-day.

It should be our endeavour to prevent the onset of shock, as carefully as we prevent the onset of sepsis. It is sounder surgery to take measures to prevent shock, than to await its onset with an armoury of remedies. He is a wiser surgeon still who will take all measures to prevent its onset, and have his armoury for its treatment ready at the same time.

Shock means a serious fall of blood pressure. This fall is brought about by exhaustion of the nerve centres. Syncope and collapse are also indicative of a fall in blood pressure—the former being a fainting due to cerebral anæmia, the latter being the condition met with in a sudden actual loss of blood, or injury to the vaso-motor system.

Surgical shock is nerve exhaustion—primarily vaso-motor, and secondarily cardiac and respiratory.

In discussing the treatment of surgical shock, one must equally refer to its prevention, and the treatment and prevention are bound up in treating and preventing the causes that produce shock. All these causes must be avoided altogether, or their effects minimised.

Both the operator and the anæsthetist must work together to prevent the onset of shock, for both of them can become powerful agents of evil in causing shock. They must consider the patient, the physique, the stamina, the nature and duration of the operation, the surroundings.

Dudley Buxton says that the theory that "the production of very profound narcosis abrogated shock, has been proved by experiment to be wholly fallacious." If, however, the anæsthesia stopped short of the third degree (*i.e.*, the stage of anæsthesia), the higher centres, though dulled, were not wholly unreceptive of impulses, and dangerous reflexes might occur—the patient being liable to fatal syncope. The deep reflexes, such as those resulting from insult to sympathetic nerves and plexuses, could not be prevented by any degree of narcosis. They could not be abolished until life was extinct. It has been shown that these reflexes, acting upon the vaso-motor centres, heart and respiration, were dangerous in proportion to the degree of narcosis after the stage of anæsthesia was established. Treatment lay in *prevention.*

Dr. Rowell, Senior Anæsthetist at Guy's Hospital, classes surgical shock under "all degrees of serious impairment of the circulation, whether produced by accidents, psychic causes, reflexis during narcosis, asphyxia, hæmorrhage, toxæmia, or exhaustion."

Too large a dose of the anæsthetic will produce shock. In deep narcosis under chloroform the blood pressure falls very markedly. This is especially marked in handling the splanchnic areas. Within wide limits, Dr. Edwards has shown that the depth of ether narcosis appeared to raise the blood pressure, showing that ether acts as a stimulant.

Chloroform is an active agent in producing shock. Ether is the safest of all the general anæsthetics for avoiding its onset. Any degree of cyanosis or impeded respiration under a general anæsthetic showed a fall of blood pressure.

Rowell also pointed out that a low blood pressure during operations induced post-operative vomiting. With a high blood pressure during an operation, there was little post-operative vomiting. The open method of ether administration tends to maintain a high blood pressure; chloroform tends to lower it.

Crile, of Cleveland, U.S.A., has fully discussed the several regions involved in anæsthetic shock. By experiment, he says that he has shown that the brain has no shock producing quality. The maximum is found in the trunk and extremities. The abdomen is more susceptible than the back, the sole and palm more than the dorsum; within the abdomen the region of the diaphragm more than the pelvis. The kidney ureters and bladder have relatively no shock values. Omentum negative, peripheral nerve trunks have high shock values. He affirms that the kind and amount of trauma inflicted by the surgeon is the single item of greatest importance. Sharp division of tissues causes least disturbance. Crushing, tearing, and dragging cause greatest amount of shock. One operator, therefore, may produce more shock in performing the same operation than another operator.

The surgeon in any operation of importance should warn his anæsthetist when he is likely to carry out some procedure which may induce shock. For instance, in nephrectomy, decapsulation of kidney, or cutting the kidney for stone, the surgeon should warn the anæsthetist when he is about to pull the kidney up into the wound. The anæsthetist is then prepared, and eases off his anæsthetic.

Fear.—The influence of fear has a decided bearing on shock. Many patients, as we all know, approach an operation in a state of terror. Fear here is a great mental factor to be combated and allayed.

Crile makes a bold statement when he says, "uncomplicated fear may cause morphological changes in the brain cells."

In certain diseases fear is a predominant and challenging factor. For instance, women suffering from exophthalmic goitre are highly nervous and mentally unstable, and unless the "fear" is here allayed, shock may be hastened and exaggerated.

Crile also says that simple surgical trauma produces morphological changes in the brain cells. Anæmia of the brain then follows, and shock follows this anæmia.

In this connection it is often a very good thing to place a nervous patient awaiting an operation beside a convalescent from a surgical operation. If this convalescent is of a cheery nature, and is happy in his or her surroundings, the effect on the nervous patient must be good.

In preventing shock during an operation, the anæsthetist is concerned in using an anæsthetic that will not greatly lower blood pressure. Here the open method of ether holds a pre-eminent position.

The warmth of the patient must be maintained on the table by having a warm table, water-bags, warm clothing, jackets for the thorax, woollen stockings for the limbs. A warm operating room; hot cloths for the open wound and its surroundings, and for the viscera hot lotions for douching and swabbing the various cavities.

Avoid giving an evacuating enema on the morning of the operation. Such an enema often seriously discomposes a waiting patient.

In severe collapse on the table, with failing pulse and respiration, stop the anæsthetic, invert the table, keep the air passages clear, open mouth with a gag, and carry out Laborde's method of rhythmic traction on the tongue. The surgeon at the same time, should, if possible, stop operating, and so prevent the inflow of dangerous reflexes.

Give pituitary extract. If the abdomen is open, pour about 2 pints of hot saline into the peritoneal cavity. The nurses should be engaged in applying hot cloths to the back of neck and epigastrium. Bandage the limbs firmly. In profound collapse and shock, carry out artificial respiration, and actual massage of the heart. Subcutaneous saline by Lane's method, or intravenous saline given slowly and with adrenalin added.

The transport of the patient from the operating-table to the stretcher, and from the stretcher to the bed, should be of the most careful nature. All unnecessary jolting should be avoided, for this tends to produce vomiting. The head of the patient before leaving the operating-room should be wrapped in a warm shawl. The body should be well covered. The bed should be warmed before with hot-water bottles and bags. The surrounding atmosphere should be warm.

All hospitals should have a small room for the reception of patients after an operation. When the dangers of shock, and the vomiting, &c. are over, then the patient can be put in the main ward. By this course, also, other patients in the ward awaiting operations will not be upset.

An inexperienced nurse sometimes makes the mistake of having the patient too thickly surrounded with hot-water bags and bottles, and of having the room too hot; this will distress the patient and induce restlessness.

What means should the surgeon employ to prevent shock? He should prepare his patient, if possible, some days previous to the operation. Prescribe a light milk diet. Gentle opening of the bowels by castor oil on the morning of the day before operation. In elderly and feeble patients give about a pint of saline by rectum three or four hours before the operation. Keep the patient free from pain. Ensure a good sleep on the night anterior to the operation, by means of veronal or heroin.

Operate early in the morning, so that after the night's rest there is no waiting.

A factor of some importance is, that the surgeon, and the patient, and the nurses are at their best in the morning. There is a diurnal variation in the pulse rate. From 3 to 5 a.m. the pulse rate is at its lowest, from 9 to 11 a.m. it is at its highest. It again rises slightly from 2 to 6 p.m., and again falls during the night. The highest temperature in the human being is in the evening. The difference between the highest and lowest pulse rate in the twenty-four hours varies from five to ten beats per minute. It is better, therefore, for the operation to be performed when the surgeon and the patient have those extra five or ten beats to the good.

For the first forty-eight hours following the operation, continuous saline should be given by rectum to all patients who are liable to exhibit shock. This should be a routine practice for all abdominal and all prolonged operations. The largest sized rubber rectal tube should be used with holes at the side, and at the end. Temperature of saline, 108° F. After 1 pint has been given, stop for an hour, but leave in the rectal tube. Then continue for another hour, and then stop. Twelve pints and more can be given in this way in the twenty-four hours. The temperature of 108° F. should be maintained, and the flow should be regulated by gravity, and not by altering the calibre of the tube. If the patient shows signs of discomfort, then the saline is too warm, or too cold, or is flowing in too quickly.

In general hospital work it is a good practice to put all operated-on patients on this treatment for two reasons:—

(1) It cannot do harm; is always a useful thing.

(2) It trains the nurses to be skilful in the method, and when they have a really bad case to do, they have the weight of experience behind them.

Heroin and atropine given one hour before the operation is useful in allaying fear and excitement, warding off shock, and making the work lighter for the anæsthetist. Some anæsthetists object to morphia at this stage on account of its influence on the pupil. Its other advantages, however, outweighs this small or doubtful disadvantage.

The atropine diminishes the amount of mucus, and by its action on the vagus nerve helps the heart.

The Trendelenberg position is valuable in preventing shock, by keeping up a high blood pressure to the nerve centres. If possible, the body should not be lowered from the Trendelenberg position till the wound is closed, and the bandage firmly applied; then the lowering should be gradual. A sudden change to the flat from this position can produce serious syncope.

Speed in operations is essential. The speedy-man—other things being equal—gets the best results. Delay increases the risk of shock. Even opening the peritoneal cavity lowers the blood pressure.

Gentle handling of viscera, and indeed of all tissues, is important. Pulling and rough handling induce shock. Viscera should not be needlessly exposed.

Continued hot cloths to the wound area is to be insisted upon. All bleeding should be rapidly controlled.

All irritating antiseptics are to be avoided. Aseptic methods are preferable to antiseptic methods.

Early diagnosis and early operations are to be our ideals. Early operating forestalls sepsis, and sepsis is a profound agent in producing shock.

Burghard recommends spinal analgesia as a means of controlling shock in severe railway and gunshot injuries, in order to prevent the passage of nerve impulses. He maintains that in this group of cases, spinal analgesia finds one of its most brilliant applications, and should be used to the exclusion of general anæsthetics.

Warnings of the approach of shock.—If occurring during the operation, it is detected by observing the wound area. The vascular oozing stops, and the cut tissues become paler. The anæsthetist will notice that the pulse is losing its volume, and that its rate increases. There is also some irregularity. The more rapid the pulse, the more profound the shock.

The condition of the reflexes here is useful in determining whether the shock is due to the anæsthetic, or the operation itself. During a long operation the pallor may be great, but a brisk corneal reflex indicates that the impending shock is not due to the anæsthetic. In shock generally the pupils are dilated, the corneal reflex is slow, the skin cold, pallor marked, and there is sweating on the forehead. These same signs indicate shock after an operation, but then in addition there is a partial return to consciousness, and very great *restlessness*.

Treatment of Shock After an Operation.

1. Rectal saline given as above.

2. Transfusion of saline into the veins. Add to this saline adrenalin (strength, 1–20,000). Let this saline flow in very slowly. Give about a pint at first, then stop, and continue at intervals slowly. The saline dilutes the blood. In shock the blood is of higher specific gravity than in health. In severe hæmorrhage, of course, the saline can be run in more copiously and more freely, as in transfusion during an operation

for a severe ruptured ectopic gestation. The rise in blood pressure following saline transfusion remains high in cases of hæmorrhage, but soon disappears in other forms of shock.

3. Warmth to the extremities, and bandage the limbs.

4. Hot cloths to the head, forehead, and back of neck.

5. Raise the foot of the bed.

6. Pituitary extract, 1 c.cm., repeated if necessary. Adrenalin, hypodermically.

7. Morphia, hypodermically; $\frac{1}{8}$ grain, repeated. Morphia is the most valuable drug at our command here, when given with pituitary extract. It raises the blood pressure and checks the dangerous restlessness. One observer has noted in many cases an immediate rise in blood pressure following the administration of morphia.

8. Avoid the use of all stimulants. Do not give alcohol. Alcohol lowers the blood pressure.

9. Do not give strychnine. Here I know that one is on debatable ground, but my own conclusions made at the bedside are that morphia is invaluable, and that all stimulants are dangerous. In collapse, however, strychnine is useful, for here the vaso-motor centres are not paralysed. Mummery points out that stimulating the exhausted nerve centres in shock may hasten a fatal issue.

10. A word about pituitary and adrenalin may not be out of place here. Pituitary will keep up blood pressure from twelve to sixteen hours. The affect of adrenalin is evanescent.

Dr. Blair Bell, who has done so much work with the pituitary gland, says that, "it acts, as indeed other pressor substances sometimes do, in raising the blood pressure better when the subject is in a condition of shock, than when in a normal condition." This drug must not take the place of saline. Both should be given.

11. In profound shock, transfuse human blood direct into the veins.

The influence of the technique of some operations on the production of shock:—

1. Exophthalmic goitres are safer under local infiltration anæsthesia (B—eucaine, adrenalin, and saline).

Neither the thyroid isthmus nor any part of the gland substance should be crushed. Cut with a knife, and stop the oozing by hot sponge pressure. Encourage the patient after the operation to drink large quantities of water. Do not pack the cavity with gauze; use large drainage tubes, and drain freely.

2. In myomectomy.—After removal of a large fibroid from the uterus, co-apt the raw uterine surfaces, but do not stitch firmly enough to produce pallor at the area involved. If very tightly stitched a very severe form of shock may be produced.

3. Crushed limbs in railway accidents.—Here spinal anæsthesia is best. If not available, infiltrate all the nerves above the site of injury, and then operate under open ether early. During the operation carry out continuous saline transfusion (with adrenalin added), or Lane's method of subcutaneous saline. Operate quickly.

4. In intussusception in children, Adams, of the London Hospital for Children, says that in twenty-three fatal cases after laparotomy and reduction, twelve died from shock, without any evidence of peritonitis at the post mortem and the degree of shock was proportionate to the length of the operation. He advocates spinal anæsthesia, for it makes the reduction easier, and allows the surgeon to make a lengthy operation even in infants. In this operation, Waterhouse keeps the child in an atmosphere considerably above room temperature. This is achieved easily in children by placing over them a cradle with electric bulbs inside, the whole being roped in with blankets. A temperature of 100°–105° F. should be maintained for at least twelve hours after the operation.

In young children the post-anæsthetic vomiting induces restlessness and shock. Here morphia is our stand-by.

5. *Gall bladder.*—I do not know if my experience is different from that of other surgeons, but I find that for this operation elaborate precautions should be taken to prevent shock. Generally these patients are bad subjects for surgery.

6. Suppurative peritonitis produces the most serious form of shock, for here there is another factor to consider—septic poisoning, with distension of the bowel. Washing out the peritoneal cavity in this operation increases the shock. Here, again, chloroform anæsthesia is very dangerous, and that continuous saline beneath the skin during the operation is so important.

7. In operations in the nasal fossæ a preliminary application of cocaine to the membranes is useful.

8. *Battlefield injuries.*—Peculiar psychical disturbances occur with some wounds. Makins reports that a man wounded over the cervical spine, and who suffered later with spinal concussion, emitted an involuntary shriek like a wounded hare on being struck. The mental state of the soldiers has an influence on the production of shock. After the battles of Graspan and Belmont, the patients came into hospital in excellent spirits. After the disaster at Magersfontein, the men were depressed and miserable. Here shock was more pronounced, and their sufferings greater. In my own experience during the South African war, shock was pronounced after severe bullet wounds with lacerations of nerves, and splintering of bone. Local shock sometimes occurred. For instance, a bullet wound of the deltoid muscle, without any nerve laceration, has produced complete loss of power in that limb, lasting for some days.

THE TREATMENT OF FRACTURES.

By R. Hamilton Russell, F.R.C.S. (Eng.).
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(Address delivered in opening the discussions on the subject in the section of Surgery.)

There are three methods of treating fractures which at the first blush appear to be very dissimilar; and they are.

First there is what I may call the old or orthodox method: this consists in bringing the fractured ends into apposition as accurately as may be, and keeping them still, while the bones unite by reparative processes analogous to those which bring about the restoration of any other wounded living tissue. It seems to me beyond question that this is, in respect of theory and underlying principle, the correct method; nevertheless experience has abundantly shown that the results obtained in cases of fracture treated in this way are often imperfect and unsatisfactory. It is, therefore, not surprising that surgeons should have striven for some improvement in their methods.

Next, there is the method of mobilisation and massage, strenuously advocated by the distinguished President of the Paris Institute of Surgery, M. Lucas Championnière, and in England by Sir William Bennett. They point out that constant immobilisation is not necessary; that the process by which bone unites differs widely from that of union by first intention as seen in the soft tissues; that callus-formation is encouraged and expedited by a little movement; that the nutrition of the muscles and the comfort of the patient are promoted by judicious frictions and massage; that subsequent stiffness of the limb is very much lessened; and that firm union is more rapidly induced. All of which beliefs are probably well-founded, except possibly the last; in any case it is of trifling importance. I would venture the opinion that in a large number of cases of fracture the practical application of this principle will furnish excellent results; also, that in a large number it will be productive of irretrievable disaster. The all important distinction between the two classes of cases has not been duly recognised and insisted upon, and we will endeavour to do this now. Briefly, in fracture of the shaft of a long bone I believe this method to be practically devoid of risk, and the result yielded is likely to be very good; but where a joint is involved, I would strongly urge that the methods of M. Lucas Championnière may be rigorously set aside. It does not seem to have been realised that the aim of treatment in the case of a fracture through the shaft of a long bone is the exact opposite of that which we seek when the injury is at the extremity of the bone and involves a joint. In the middle of the shaft we desire an abundant production of callus; we can hardly have too much; where the fracture involves the joint, however, we cannot possibly have too little. In the middle of the shaft callus is required to surround the ends of the bones, to fill in all the interstices left by imperfection of apposition, and hold the fragments firmly together until union is consolidated; our sole object is complete and permanent fixity. In the joint fracture none of these requisites are present; there is little or no fear of non-union; the whole aim of surgical endeavour will be directed towards conducting the case in such a way that no stiffness of the joint shall result. There are two chief causes of stiffening of a joint after injury, a lesser and a greater cause. The lesser cause consists in adhesions due to traumatic arthritis; the greater cause, overwhelming in importance, in its capacity for evil and its intractibility, is excessive and unnecessary callus formation. We may practically regard the question of arthritic adhesions as trivial; those that result from simple non-infective traumatic arthritis are never productive of serious or permanent trouble. But superfluous callus is a very different matter. If it projects on to the articular surfaces, it will be destructive of the integrity of the joint; any attempt at movement will then cause a fresh trauma and aggravate the arthritis. In a compound joint such as the carpus, it will weld the bones together, and it will involve the bony grooves of tendons, the tendon sheaths and the tendons themselves in a common ruin. I cannot too strongly emphasise the fact that excessive callus-formation among joint structures, with the lamentable results that I have detailed, can be very readily induced by early mobilisation of the joint; further that it can be brought about in no other way except by too early mobilisation of the joint. On the other hand I would urge with equal insistence that immobilisation of the joint *per se* is never a cause of permanent stiffness, and that it cannot be too complete, or even, within ordinary limits of reason, too prolonged, in cases of joint fracture. Of course I am well aware that after prolonged immobilisation a joint will appear at first somewhat stiff owing to rigidity of the muscles from prolonged inaction; but this is a form of stiffness which is quite devoid of serious import, and if treated by masterly inactivity on the part of the surgeon will always be transient.

The foregoing remarks do not apply in any case in which there is fracture through the joint surfaces with displacement which cannot be rectified by manipulation, *e.g.*, in fracture through the lower humeral epiphysis, where a portion of the epiphysis is separated and twisted out of place. Such an event constitutes a special indication for immediate operation in any case, and does not come within the scope of my present argument. What I have said about the danger of early mobilisation in cases of joint fractures, and the safety and eminently beneficial effect of fearless and uncompromising immobilisation of such injuries, is at variance with the views held by a large number of surgeons. There is a very general and, as it seems to me, ill-considered notion that in any case of injury to a joint where stiffness is threatened, the right way to anticipate and prevent such stiffness is to keep the joint on the move. I have even heard of such a thing as a child being chloroformed every other day, in order that passive movement might be carried out in a case of injury to the elbow. I can only hope that such a proceeding as this is of very rare and exceptional occurrence.

I was greatly struck some years ago, when I was on the active staff of the Melbourne Children's Hospital, by observation of the behaviour of fractures involving joints, and of the methods that furnished the best results. I can best indicate the nature of these observations by stating the following laws which I formulated for myself, relative to the management of such cases:—

(1) In fracture involving or in the immediate vicinity of a joint, where there is no gross displacement of the articular surfaces, stiffness of the joint never supervenes, except as the result of passive motion carried out by the surgeon.

(2) In fractures involving joints in which there is some displacement or irregularity of the joint surfaces, and in which, therefore, some impairment of mobility is inevitable, such stiffness will be greatly aggravated by the employment of passive movement, and complete abstention from passive movement by the surgeon will yield the best result attainable.

(3) When great stiffness is present in spite of the surgeon's efforts to increase the mobility by passive movement, such stiffness will persist so long as the surgeon persists.

(4) When the surgeon abandons the case in despair, improvement will at once commence.

Condensing these four laws into one weighty generalisation we may say this: In fractures involving joints the most potent cause of stiffness is the so-called passive movement performed by the surgeon. Immobilisation is never a cause of enduring stiffness. The maximum degree of permanent usefulness is to be obtained by immobilisation alone.

There are few surgical rules of more insistent value than this strange seeming paradox that immobilisation makes for good movement in a joint, and that over-zealous mobilisation stiffens; moreover there are few that are more likely to be lost sight of, disregarded, or positively disbelieved.

For many years now, passive movement of a joint for the prevention of stiffness resulting from a fracture in its neighbourhood has been unknown in my work. Whatever may be the cause operating in any individual case, one thing appears to me to be certain: if we try to overcome the disability by force, by what is called "passive motion," we shall make matters worse instead of better. The arthritis, if there be arthritis, will be aggravated; if there be not arthritis, it will be created. If the tendons or muscles be involved in callus or cicatricial tissue, forced movement will be productive of more callus and more cicatricial tissue. The way to control the formation of callus, to lessen the amount and density of the cicatricial tissue, and to mitigate the severity of the arthritis is to immobilise the joint; and the result of our action in immobilising the joint will be to shortly enable the patient himself to mobilise it.

Let us now turn to the consideration of the third method of treating fractures, that which is associated with the name of Mr. Arbuthnot Lane, who advocates an almost universal, or at any rate a very free and ready resort to open operation. Briefly he considers that the correct way of securing the best result is to expose the fragments and fasten them together with a metallic plate and screws, or with some such appliance.

Needless to say there can be only one possible plea for the general adoption of Mr. Lane's principle, which is, that good results cannot be obtained by any means short of operation; and this is the plea that Mr. Lane advances. Our confidence in accepting this principle is shaken at the outset by the fact that M. Lucas Championnière, Sir William Bennett, and many others, are perfectly and apparently justly satisfied with the results they are able to obtain by non-operative methods. I see no reason to doubt, judging from the published accounts, that the results obtained by those distinguished surgeons are as good as those obtained by Mr. Lane; nor on the other hand can I doubt that Mr. Lane is quite as capable as they of obtaining good results by non-operative methods, were he to try hard enough.

When, further, we come to consider the reasoning upon which Mr. Lane bases his position, we are not reassured. Briefly stated, it amounts to this, that he has investigated the final results of fractures treated in a large London hospital during some years, and he has found that many have been unsatisfactory or bad, whereas the results that have accrued from operative treatment in his hands have been uniformly very good; from this the inference is drawn that operation is the right treatment. I do not dispute the facts; but I reject the inference. The argument appears to me quite unfair. I think it will not be disputed that cases of fracture in a large hospital receive very much less of the individual attention of the surgeon-in-charge than any other class of cases. When a fracture comes in, it is admitted and put up by the house-surgeon, and the chief will often take little or no part in the treatment unless specially asked to do so by the house-surgeon, because his time and attention are very fully engrossed by cases that seem to be of greater urgency. Now, the house-surgeon, no matter how capable, industrious, and painstaking he may be, and usually is, is nevertheless at the threshold of his experience in the practical work of surgery. Moreover, at the end of every six months, when he has acquired considerable practical skill and experience, he goes and makes way for another who has the skill yet to acquire; and in reviewing his six months tenure of office he will, being a reflective and observant man, probably make some such observation as this:—" If there is one thing that more than another has been impressed upon me by my experience as a house-surgeon, it is that fractures are the very dickens ! " It is clearly unsound to contrast the results obtained by a long and varying series of surgeons, alike only in one common feature, viz., that they are all in their novitiate, with a similar series that have passed through the hands of a single surgeon of the mature experience and practised skill of Mr. Lane, for it goes without saying that the cases operated upon by him receive a very full measure of his personal supervision. The only fair comparison would be afforded by a series of cases treated by a single surgeon comparable to Mr. Lane, and such a condition seems to be provided by the experiences of such surgeons as M. Lucas Championnière, Sir William Bennett, and many others, who have expended upon the non-operative treatment of fractures as much consideration and pains as Mr. Lane has bestowed upon operative methods. I believe the result of this comparison would be to establish the conviction that operative methods are not so essential to the securing of good results as Mr. Lane thinks they are. Again, if we consider closely what is really accomplished by operating on a fracture, there does not seem to be quite so much in it as would at first appear. It is something, of course, to be certain that no soft tissues intervene between the fragments, and it is much to render it impossible for the bones to overlap and produce shortening; and it is convenient to have the bones firmly held in position while the splints are being applied. But the plate and screws or the wire have really nothing to do directly with the processes of repair, and their presence, however skilfully inserted, will not do away with the need for care in the after treatment, nor prove in themselves a trustworthy security against bad result.

There is another aspect of this question of operation on recent fractures that I would state with great seriousness. The point is not merely as to which is the best and simplest method of obtaining an ideal result in the case of any individual patient who chances to come under the care of an experienced operating surgeon in a perfectly appointed hospital with every facility to hand. The treatment of fractures is the one difficult branch of surgery that must be undertaken by the busy general practitioner with few opportunities for operative practice, with perhaps little taste for it, under all kinds of disadvantageous and trying conditions. With this in view, what course are we going to recommend? Are we to urge a very frequent and ready resort to operation as the solution of every little difficulty that arises in managing a fracture, or are we going to strive for means of amending and improving our non-operative methods, seeking out and ear-marking the causes of failure and the conditions of success, recognising that excellent results can usually be attained by judicious and skilful management. The latter course seems to me the right one, and I cannot regard without apprehension the increasing trend of surgical practice in our large hospitals towards frequent open operation in cases of recent fracture. Let me cite an instance in illustration of my position on this question. A month or two ago my house-surgeon found great difficulty in the management of an oblique supra-condylar fracture of the femur in a middle-aged woman. On examining it with him I quite agreed with his views as to the difficulties presented, and decided to operate by Lane's method, pointing out that this was in my opinion one of the exceptional cases in which operation is indicated. Then I bethought me of my students, and decided that I would do nothing of the kind. They were there to learn all I could teach them about surgical principles and the management of difficult fractures. So we set to work, and, with great pains and frequent readjustments of our apparatus, in the course of a few days we got the fragments into good position and the limb perfect in length. Both they and I learnt a great deal, and I venture to think those lads could treat a bad fracture of the thigh with many of us. When they go out into practice the principles they learnt then will avail them much, not only in treating fractures of the thigh, but in treating other fractures as well. Should they meet with such a case, in circumstances under which operation would be impracticable or most unwise, they will know not only that it can be successfully treated without operation, but also how to set about doing it. Whereas, had I performed an operation they would not have gained that valuable piece of surgical equipment, and they might some day have been placed in a position of disadvantage through my fault. Briefly, it seems quite clear to me that unless hospital surgeons and teachers are prepared to advocate that open operation on recent fractures should be undertaken by all and sundry under all sorts of conditions, they must let it be seen that they themselves regard non-operative methods as of paramount value and importance; that they shrink from resorting to open operation in any but quite exceptional and rare instances; and that they frankly acknowledge that the necessity for treating a fracture by open operation carries with it in some measure, greater or less, an adverse reflection upon their own skill. Unless they do this how can the general practitioner who goes out from our medical schools be properly trained in this the most important branch of surgical work he will be called upon to undertake. Obviously he cannot.

And now I am conscious that, while I have admitted that the result of treatment of fractures in the past have been often bad, I may seem to be opposing any change in our methods without offering even a helpful suggestion. Let me try to do better than this; and to begin with, I would say that much that is of value is to be learnt from the methods of M. Lucas Championnière; what is called treatment by mobilisation and massage. The first great fact to be learnt is that uniformly good results can undoubtedly be obtained by non-operative methods; and if they can be they ought to be. Granted that an operation be safe, that is not in itself a sufficient reason for doing it; for the performing of operations is not the first duty of the surgeon, but the last. The mobilisation and massage method, however, excellent as it has proved in the hands of its distinguished advocate, is in my opinion quite as unsuited for universal employment as open operation. But I have a strong suspicion that the moving and the massage have really very little to do with the excellence of the results obtained, and that the main factor of success is in reality the frequency with which the apparatus has to be taken off and reapplied. I would suggest that here we have a hint of great value. I have been impressed with the fact that bad results are frequently caused through a fracture that has been put up in splints or plaster of Paris being left for too long a period without inspection, because all appears well on the outside, and the patient expresses himself as comfortable; meanwhile the fragments may have gradually become misplaced, and the apparatus loose and ineffective. It is of course a cardinal rule in the treatment of fractures that splints shall be taken down and re-applied in the first twenty-four hours; but to this I would suggest in addition that they may be taken off frequently and the fracture examined again and again during the first week or two, so that defects of position may be recognised and righted during the early days. If this were done systematically I am inclined to the belief that the treatment of fractures would prove far more satisfactory and less difficult than it is at present reputed to be; and that the shadow of a reproach which I, for one, feel to be perilously near to us will in natural course recede and pass completely away.

THE TREATMENT OF FRACTURES.

H. S. Newland, M.S., F.R.C.S., Adelaide.

In the treatment of fractures our profession has too long been hampered by tradition. The time is now ripe for us to take stock of our methods and to see whether they cannot be improved upon. The Committee of this section have acted wisely in selecting this subject for discussion. An important investigation of the same question is at present being conducted in England, and should lead to valuable results. It is a truism that without an accurate diagnosis correct treatment is impossible. Every means at our disposal should therefore be employed in order to elucidate every feature of a fracture. It was not so very long ago that it was considered rather a reproach not to be able to diagnose and treat any form of fracture without the use of the X-rays. Few, I hope, are of this opinion to-day. I do not deny that perfect results may be obtained without the rontgen rays in an enormous majority of fractures. Nevertheless, skiagraphy is the only method of examining a fracture which clears up in every detail the actual injury to the bone. Please observe that I say "the actual injury to the bone." It does no more than this. Concomitant injury of the soft parts is detected in other ways. Not only does skiagraphy confirm and amplify the obvious, but reveals injuries to bone which may be unsuspected, or, if suspected, incapable of proof.

Fissured fractures of the long bones, a transverse fracture of the patella, a fracture of the process of the ulna, of the carpal scaphoid, of the base of the first metacarpal, all these fractures and many more can only be certainly diagnosed by skiagraphy. Further, in sprains and dislocations complicated by injury of the bones, skiagraphy alone can reveal the exact nature of the bony lesion.

Skiagraphy does more than make plain to us the precise injury to the bone or bones. It explains in some cases how the injury is brought about. The spiral fracture so frequently seen in the shaft of the tibia is obviously due to torsion. Skiagraphy also furnishes valuable indications for treatment. It has been shown by Reed and others that in nearly every fracture of the shaft of the radius, the upper fragment of the radius is supinated. This fact was detected by observing the position of the tubercle of the radius. It is obvious that in every case the fracture should be put up in a position of supination. Were this always done I believe we should see fewer of those cases suffering from deficient pronation and supination. To effectually maintain the forearm in a position of supination it is necessary to have a posterior gutter splint of some malleable or mouldable material or of plaster of Paris extending from the heads of the metacarpal bones half way up the arms. The elbow is flexed to a right angle.

In Colles fracture I do not employ a splint. The deformity does not recur if it has been properly reduced. To reduce the displaced lower fragment of the radius firm traction should be made on the hand, which should then be fully flexed at the wrist. This position is maintained in the following ways. A piece of 20 strapping is carried along the radial side of the forearm and across the back of the hand at the level of the bases of the metacarpal bones and then up along the ulnar side of the forearm. This position has two advantages, The stretched extensor tendons which groove the back of the radius prevent backward displacement of the lower radial fragment, and secondly, the patient recovers with a full measure of flexion. It is flexion of the wrist which is usually limited after a Colles fracture. The strapping is removed daily and the wrist and forearm massaged, and active movements of the fingers and wrist encouraged.

I do not employ a splint in the case of fractures of the lower end of the humerus. The limb is put up in the Jones position. That is, the elbow is fully flexed, if necessary under an anæsthetic, and kept so with a piece of 20 strapping passed round the middle of the arm and forearm. Massage is started from the beginning. In a few days the strapping is removed daily, and massage, passive and active movements, performed.

Treated in this way most cases do admirably. The flexion certainly corrects the backward displacement (radiography proves this), but it also prevents the elbow from becoming stiff in a position which might considerably limit flexion.

It should never be forgotten that our efforts to get a broken bone to unite are simply taken as a means to an end. That end should be the restoration of function. This is too often not borne in mind. I frequently see patients at the O.P. Department whose bones have united perfectly, but whose soft tissues are wasted and adjacent joints more or less stiff. It cannot be too strongly insisted that our whole aim must be to get restoration of function and nothing less. To obtain this ideal it is not necessary, and even if it were necessary it is not possible, to get the fragments of the broken bone into position, so that the bone is restored to its original form. It suffices if the alignment is good and the overlap is as far as possible corrected. Consequently I am not an advocate of the open method of treatment of fractures of the shafts of the long bone. I believe that in some cases of fracture in the region of the trochanters of the femur an operation might be performed with advantage. In some of my skiagraphs the displacement has been extreme with much eventual shortening. Open operation is generally demanded when a fracture is complicated by involvement of a nerve, by pressure on the spinal cord or by intracranial tension. It is not the injured bone which is demanding operation. In ununited fracture there is of course a legitimate field for operative surgery.

AN OPERATION FOR SEVERE FORMS OF HARE-LIP.

H. S. Newland, M.S., F.R.C.S., Adelaide.

About a year ago, in Sir William Stokes' "Operative and Clinical Surgery," I came across a method of treating hare-lip, which he had practised in three cases. The following is a condensed account of the operative procedure he followed, given mostly in his own words:—"Having either removed the premaxillary bone, should it be displaced, or placed it in its normal position, I then with a curved scissors divide all the false frena. I seize the edge of the cleft at its lower extremity with a double hook, draw the parts forward in order to put them on the stretch, and with a fine narrow-bladed knife, I make an incision through the entire thickness of the lip, taking care, however, to stop about two or three lines above the red border of the lip. I then make a similar incision on the opposite side. Two serres fines must then be applied to the extremities of these flaps, which have the twofold advantage of arresting any hæmorrhage that may occur, and also by their own weight making the flaps hang down below the red border of the lip, thus keeping them out of the way of the operator until the time comes for uniting them by a point or points of interrupted suture. The two points can thus be brought down below the red border of the lip, thus forming a projection and effectually getting rid of the subsequent occurrence of a notch in this situation. The next step consists in making a vertical incision through the entire thickness of the lip with a small straight scissors at the upper angle of the cleft. This, of course, is only necessary in cases where the labial cleft does not extend into the nose. The making of the partial incisions at the edge of the cleft on either side constitutes the third stage of the operation. These incisions are made through fully two-thirds or three-fourths of the thickness of the lip. Great care should be taken to prevent the knife going too far and wounding the mucous membrane of the lip. The two quadrilateral flaps united behind by the mucous membrane of the posterior aspect of the lip can then with great facility be turned back, and the broad raw surfaces of each brought into apposition." I need not refer to the manner in which coaptation is brought about. It is now out of date. The idea of closing the cleft without removal of tissue by paring strongly appealed to me, and when, soon afterwards, a case of severe double hare-lip came under my care at the Children's Hospital, I resolved to put it into practice. Besides the complete or bi-lateral hare-lip and cleft palate, the premaxilla and prolabium were very prominent.

The Operation—Stage I.—The reduction of the premaxilla. Removal of the bone should never be performed. An incision is made in an antero-posterior direction along the lower border of the vomer anteriorly. The mucous membrane is separated on each side from the vomer, which usually has a bulbous expansion at this spot. Killian's instruments are used for this purpose. A V-shaped piece of the septum is removed and the premaxilla can then easily be pressed backwards between the maxillæ. The mucous membrane on each side of the premaxilla and at the anterior end of each alveolar process of each maxilla is pared, and the premaxilla fixed to the maxilla on each side with a silk-worm gut stitch.

Stage II.—A traction stitch is passed through the lower end of the prolabium, and one on each side through the lateral margins of the clefts. Drawing on these renders the subsequent manipulations much easier of performance. A V-shaped incision is now made in the prolabium, the limbs of the V being placed along the muco-cutaneous junction, and the incision extending in depth as far as the mucous membrane on its under surface. The lateral margins of the cleft are next dealt with. A knife is thrust through the whole thickness of the lip at the lower extremity of each lateral margin, and an incision made in a upward and inward direction. An incision is now carried from the cut just made upwards along the muco-cutaneous margin, into the nostril. This incision extends in depth to the mucous membrane, but must not involve it.

Stage III—Introduction of sutures.—The mucous membrane turned back from the central and lateral margins of the clefts is sutured with catgut continuous or interrupted. The large raw surfaces bounding the clefts are next apposed with silk-worm gut, the finer the better. Those portions of the lip cut by transfixion are sutured in the mid-line below the prolabium. If there is any tension, and there not infrequently is some just below the prolabium, any false frena should be divided from the facial surface of the maxilla.

Stage IV—Insertion of tension stitch.—All tension on the line of union is avoided by passing a silk stitch from the mucous aspect, through the thickness of the lip. The needle is introduced at the spot on the skin from which it emerged and passed across in the substance of the sutured lip through the skin. Again it is made to enter its point of exit and finally emerges through the mucous membrane on the deep surface of the lip, when the ends are tied together. No dressing is employed. Any crusts of blood or discharge blocking the nostrils are removed as they form, and a little sterile vaseline applied. The sutures are removed on third or fourth day.

Results.—I have performed this operation on three of these severe cases, and have obtained an excellent result in all. There is no notch in the upper lip, and no groove in the line of the cicatrix. It is applicable to all forms of hare-lip, and will, I believe, yield results superior to those obtained in cases upon which one or other of the paring operations has been performed.

IODOFORM BONE-PLUGGING IN CHRONIC OSTEOMYELITIS AND TUBERCULAR DISEASE OF BONES.

By T. F. Ryan, M.B., C.B.
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In the treatment of bone cavities caused by tuberculosis or chronic osteomyelitis, I think the treatment originated by Professor Moorhof, of Vienna, supersedes all other forms of treating these conditions. This method is that of iodoform bone-plugging, and the mixture recommended by Moorhof consists of the finest pulv. iodoform and oil of sesame, sixty parts and forty parts each of spermaceti; this mass becomes fluid at a temperature of 112° F., and hard at the temperature of an ordinary room. Instead of using pulv. iodoform, I use the crystal of iodoform, and have them sterilised for two days in a solution of carbolic acid; it is then dried in an oven and remains covered till required. I do not use oil of sesame, and consider that the iodoform and spermaceti are as efficacious without it. I melt the spermaceti in a porcelain vessel with a spout and handle on it. The iodoform is put in while the spermaceti is fluid, and the whole mixture is kept stirred by the nurse with a teaspoon. The vessel containing the paste stands in a receptacle containing hot water at a temperature of 120° F.

The instruments I have used in the operation are ordinary carpenter's chisel and a mallet. No Esmarch bandage is used, and the limb is not elevated before operation. The scar tissue surrounding the sinuses is removed by elliptical incisions, the periosteum is peeled back with elevators, and the mallet and chisel are used freely to cut a trough-like cavity with nearly vertical sides. All diseased bone is removed until the cavity is tolerably clean. Large burrs attached to an electrically-driven motor are then used to make a smooth even hard surface. The motor lessens excessive bleeding, which, however, never causes me any trouble. The cavity is then mopped out with pure carbolic acid, which is washed away with very hot water. In my first case I used adrenalin chloride, but discontinued it later. The cavity is next dried with hot air by means of an electric apparatus. The packing mixture is now poured into the cavity, and it is necessary to see that it reaches all the parts of the cavity and filled flush with the opening in the bone. At this stage it may be necessary to use a metal spatula to fashion the superficial surface. In a few minutes the mass hardens and the periosteum can now be stitched with cat-gut, the skin and underlying structure united with silk-worm gut sutures, and the limb put on a poroplastic splint. No drainage material is used.

In my cases there has been no rise of temperature, and the three cases have healed by first intention. In one case where a cavity in the femur 7 in. long x 1½ in. deep was filled with the mixture, there was a very small percentage of iodine excreted in the urine for seven days, when it then disappeared.

Absorption can only take place from the peripheral portions of the plug, and this must be small in amount. In my first case, tubercular disease of the right ulna, the case had been treated for two years elsewhere by scraping and chiselling, but the sinus still remained. The cavity was prepared in the way I have indicated, and was about 2 in. long, ¾ in. broad, and an inch deep when all diseased tissue was chiselled and burred.

It was sewn up without drainage, and the patient, who was a house-painter, was hard at work in sixteen days. The skiagraph shows the filling remaining at that time, the other skiagraphs show the gradual absorption of the plug, and its final disappearance three months after operation, and its replacement by osseous tissue. In all the skiagraphs after operation there will be seen a halo of granulation tissue round the plug—this eventually becomes osseous.

The skiagraph of the femur is one of chronic osteomyelitis, and in the case the trough produced after chiselling and burring was 7 in. long, 1½ in. deep, and 1¼ in. wide, and embraced about two-thirds the length of the femur. It was filled with the crystalline iodoform and spermaceti, and the whole wound healed by first intention without any rise in the temperature. The other case was one of tubercular disease of the tibia, having a sinus leading to the cavity of two years' duration. This cavity when prepared was 5 in. long by 1½ in. wide, and healed by first intention.

To ensure the success of the operation one must excise infected skin freely, peel back the periosteum, remove all bone anterior to the cavity. This is best done with a sharp carpenter's chisel, ½ inch in width, and a strong copper mallet. A large burr rapidly driven to make the wall smooth, and hot air to make them dry. It is very necessary also to clip the edge of the periosteum with gauze, otherwise hardened portions of the filling may become scattered through the soft tissues. In any case this makes no difference in its absorption. The treatment is expeditious, and the opportunities for its application occur in the practice of most medical men. It permits the patient to follow his usual work in a short time, as compared with the usual operative methods of treating bone cavities, the result of tubercular disease or chronic osteomyelitis.

OPEN ETHYL CHLORIDE ANÆSTHESIA FOR MINOR OPERATIONS AND AS PRELIMINARY TO OPEN ANÆSTHESIA.

By R. W. Hornabrook, M.B., C.B. (Adelaide), M.R.C.S., L.R.C.P. (Eng.).

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In reviewing nearly 2,000 cases of open ethyl chloride anæsthesia for dental and minor surgical operations, the question one naturally asks is, What are the advantages and disadvantages of this method? The cases in which open ethyl chloride has been administered during the past eighteen months are mainly extraction of teeth, removal of post-nasal growths, tonsillotomy, removal of aural polypi, opening of abscesses, a few cases of setting of fractured limbs, and reduction of dislocations. Of the above cases, nearly half have been dental.

The principal reason that led the writer to adopt the open method of administration, was the extreme unpleasantness of the closed method from the patient's standpoint, and also the feeling that the patient often has that the closed inhaler, especially if it has a rubber mouthpiece, is unclean. It does not matter how careful or expert the anæsthetist may be, there is the impression that the rubber mouth, or any closed inhaler, has been used by hundreds of persons before, some of whose conditions have not been at all sanitary or pleasant. There is also the fear with the closed inhaler that there will come over the patient a feeling of suffocation—a fear which I am sorry to say is not at all unwarranted. These were, however, not the only reasons which force me to advocate the open method of anæsthesia.

I am not an apostle of the closed method being even the most scientific method, nor an advocate that even a moderate amount of asphyxiation is of benefit to the patient, in that raising of blood-pressure by means of CO_2 assists the surgeon or operator by maintaining his patient in a more fit condition to resist the shock of his operation, nor do I believe that the sudden throwing into the system of a concentrated dose of a powerful anæsthetic, such as very often occurs when ethyl chloride is used by the closed method, is of benefit in any way, but I do believe that it is extremely dangerous. It requires no more than ordinary common sense to see that when an anæsthetic is administered, there is a strain out of the ordinary thrown on the nervous and circulating systems. Is it right that this should be brought about by a greater shock than necessary—by the inhalation of a concentrated dose of anæsthetic plus the absence of free supply of air, and the harmful results of re-breathing the poisonous products of one's own breath?

In the open method of ethyl chloride administration, the drug enters the system gradually and with free admixture of air, the shock given is more in the nature of a gentle push into unconsciousness as against the more violent and rapid rush into a state of insensibility in the closed method, and with the open method there is no feeling of suffocation or desire to fight for air. It has been very noticeable that there is very seldom any headache or feeling of great fulness in the head after the open method, both of which conditions are common after the closed. Ethyl chloride is very rapidly eliminated from the system, so that a few minutes after the completion of the operation the patient is quite fit to go home, a big advantage from the patient's standpoint as against ether or chloroform. Vomiting is rare unless the patient has swallowed some blood, in which case it will be brought up, but the feeling of sickness soon passes away. In the previous sentence I wrote, *swallowed* some blood, and this is very noticeable in dealing with open ethyl chloride, that the swallowing and coughing reflexes are retained, a big point when dealing with post-nasal and tonsillotomy cases, and also with dental extractions, for here, if any blood gets into the back of the throat, it is swallowed, or trickles into the larynx, a cough is at once set up, so that the fragment of post-nasal growth or other foreign body is thrown up. On numerous occasions, after the extraction of a number of teeth, I have allowed the patient to come to, sitting up in the dental chair, and not putting his head over the spittoon—directly the blood gets to the back of the throat a reflex cough has been set up, as one so often experiences when a small pellet of phlegm tickles the throat.

Given the advantages of open ethyl chloride, what are its disadvantages? One is, it is a slightly more expensive method of administering the drug, 8 to 10 c.c. being used in the majority of cases as against 3 to 5 c.c. by the closed method. A No. 70 tube of Kelene graduated to 50 c.c. should do for six or seven administrations. Another disadvantage is that, in the cold weather especially, a patient is occasionally met who is difficult to put deeply under the drug. He will go to a certain stage, but on attempting to extract the teeth, his hand will be raised or he will make some movement that interferes with the operator. The class of case in which this may occur is that of the full-blooded male; the fault does not, however, rest entirely with the drug, but may be due to the ethyl chloride being sprayed too much on to one spot and not over a fairly wide area, which increases the evaporation. In the majority of cases, where the administrator meets with incomplete success, the fault lies in some failure of technique. A good method of administration in cases of this sort is to put the patient off with open ethyl chloride and then to deepen the anæsthesia with closed somnoform. By this means the struggling stage which may be met with is generally avoided.

The length of time that a patient remains quiet after being anæsthetised by open ethyl chloride is from one to one and a half minutes, that is, if the anæsthetic is discontinued, such as is the case with dental extractions and removal of adenoids. Open ethyl chloride can, however, be administered for a longer operation, such as removal of aural polypi, or setting a fractured limb. I have had a patient under for twenty minutes by the open method, but I do not recommend it for cases of more than five or ten minutes' duration, as it would be very expensive and has no advantage over open ether when prolonged anæsthesia is required.

What are the advantages of open ethyl chloride as against somnoform by the closed method? Firstly, I think it is certainly much safer. In the whole of my experience of open ethyl chloride, either by itself or as a preliminary to open ether anæsthesia—that is, in nearly 3,500 cases of all ages, both sexes, and nearly every major or minor operation—I have not once had a moment's anxiety or had to resort to any artificial means of restoration. I think this fact is mainly due to absence of sudden concentration by the open method, and the feeling in the patient's mind that there is nothing to struggle against—he knows that he will not be suffocated, but will get air the whole time. In dealing with plethoric men I certainly think it is easier to put them under with somnoform entirely, or to follow the open ethyl chloride by closed somnoform—this is especially the case in the cold atmosphere of winter. If you start with somnoform by the closed method, do not rush it, but bring the mask gradually closer to the patient's face—no good is ever done by rushing an anæsthetic; even with nitrous oxide administration, the air supply ought to be cut off gradually, if necessary.

Ethyl chloride is certainly a more pleasant drug for the patient than somnoform, though the present formula for somnoform contains 83 per cent. chloride of ethyl as against 65 per cent. in the original. The sensation of floating off into unconsciousness by means of open ethyl chloride is a most delightful one, especially if the administrator does not try to rush the drug at the start. With open ethyl chloride the conjunctival reflex is lost early; this is followed by the breathing of the patient becoming of a stertorous or snoring character, especially if the mouth has not been gagged wide open. When the breathing takes on this vibratory character, which is generally within one and a half or two minutes from the very commencement of administration, the patient is ready for operation of short duration, such as the extraction of two or three teeth, opening of an abscess, or removal of adenoids by an expert operator. The whole of this time the corneal reflex is well marked. Should the administrator wish to deepen the anæsthesia, he may continue the administration for ten or a dozen breaths after the vibratory character of the breathing has commenced; by this means the patient remains a short time longer under the influence of the drug and more time is given the operator, but even in these cases the corneal reflex is not lost, except in rare instances. In fact, with open ethyl chloride, one of its great charms and points of safety lies in the fact that the corneal, swallowing and coughing reflexes are retained.

With closed somnoform, on the other hand, the loss of the corneal reflex follows very closely on the breathing taking on a stertorous character. The period of quiescent anæsthesia with open ethyl chloride lasts, as a general rule, from one to two minutes; this is followed by a stage in which, if the operator still continues with his work, the patient may call out or raise his hand. These acts are, however, entirely voluntary, the patient knows nothing of them, and if asked after the operation will deny that he did so or say he did not know he struggled. Following on this stage is one in which the patient seems to be quite awake—he can hear you talking and can remember what you say to him, and if told to open his mouth or shut it, or raise his hand, will do so, but during this stage there is well marked analgesia. In several cases this has been well shown at the Melbourne Dental Hospital—a patient has come round or reached the struggling stage before all his six or seven teeth have been removed. Instead of giving him a second administration, which can be done if necessary, without any apparent risk, I have allowed him to come round and told him to open his mouth and one or more remaining teeth have been taken out without trouble or pain.

In a recent case at the Eye and Ear Hospital the house surgeon was unable to remove a nasal polyp from the left nostril before the patient came to. I gave the patient, a lad of 14 years of age, a second dose without any objection on his part; and this is very noticeable about open ethyl chloride administration, it is very rarely indeed that the patient objects to a repeated dose. Again the house surgeon tried, but failed. I then allowed the patient to come to, and asked the honorary surgeon if he would mind removing the growth, as the lad would feel nothing. The nose was swabbed out with cotton wool and the snare again inserted and the growth this time removed, the boy feeling no pain at all.

In all my open ethyl chloride administrations the head and shoulders are kept raised up either on a pillow, or the movable headrest is raised; the pillow can be removed or the headrest lowered, if the surgeon wishes it, whilst operating. In my dental cases at the

Melbourne Dental Hospital, the patients sit upright in a chair; there has never been any cases of collapse. I am not a believer that the head low is the safest method of administering an anæsthetic; with the head up, in a natural position of sleep, the anæsthesia of the patient comes on easier and earlier than would be the case if the head is low and the brain congested, and, moreover, should collapse occur, it will come on when less anæsthetic has been taken into the system; and moreover, the anæsthetist who keeps his patient's head up has something to fall back upon, for by lowering the head he can increase the flow of blood to the brain and thus stimulate his cardiac and respiratory centres.

In dealing with dental work and the closed method of somnoform or ethyl chloride administration generally used by a dentist, I have been struck by the few deaths that have occurred in spite of the upright position generally assumed and in spite of the fact that scares or collapses are fairly frequent when the closed method is used, and I am of opinion that this freedom on the part of dentists from fatalities is largely due to the fact that the drug was administered in what is generally looked upon as a dangerous position for the patient—namely, the upright or semi-upright.

This freedom from fatalities is, I think, due to anæsthesia coming on earlier than would be the case with full congested brain, and also the fact that when the patient does collapse the rush of blood, when the head is lowered, causes a stimulation of the cardiac and respiratory centres, and less anæsthetic having been taken into the system than would be the case if the patient had been anæsthetised lying down, the patient is able to respond easier to that stimulation.

One final word as regards the use of the gag in dental or adenoid cases. I do not open the gag before starting my administration, but place it in the mouth, telling the patient not to bite on it, but to leave it lying loose in the mouth—in fact, to try and forget it is there. With young children, if at all frightened, I make no attempt to insert the gag until after they are fully under the anæsthetic. If the patient has followed instructions the gag will open easily, but if the patient has bitten hard on it whilst going under, there will be a certain amount of difficulty in first trying to open the mouth owing to rigidity.

I have administered open ethyl chloride on several occasions to myself, starting when I was in a standing position, and, as I felt the influence of the drug overpowering me, lying down. The sensation of going under is that of a dreamy, floating away, and then oblivion, followed by a slow return to consciousness, with no headache or after effects.

I hope, before the completion of this Congress, to be able to demonstrate the ease, simplicity, and safety of the method, for if a man attempts it on his own before he has seen the method or been shown it, his attempt will most likely end in failure.

1. For minor operations of short duration, where open ethyl chloride is used by itself, the best material to use is honeycomb towelling of medium thickness, and of one layer only.

2. For prolonged operations when open ethyl chloride is followed by open ether, use a mask covered with one layer of flannel medium thickness, two layers of domette, or eight layers of white gauze.

3. In the open ether anæsthesia that has been preceded by open ethyl chloride, bridge over the stage between the two with a few drops of chloroform.

4. *Lint*, as a material for covering mask, is strongly objected to by the writer, owing to the thickness of the material, and the vapour of condensation forming on the inner layer of mask and the danger of shutting in a 2 per cent. or more of chloroform vapour, even when only a little chloroform has been dropped on at a time.

5. The best make of ethyl chloride to use is that known as Kelene, not that the ethyl chloride is better than other makes, but the preparation is got up in 50 c.c. bottles, with a patent stopper, which makes it very easy to handle. Kelene has also a pleasant odour, which some of the other preparations have not.

6. In using Kelene from the No. 70 bottle containing 50 c.c., be careful not to spray any of it into the patient's eyes, as it is very painful.

7. In order to get a free drop and prevent spraying, insert a very small piece of cotton wool under the stopper—it will then be found that on gently elevating the lever the Kelene will drop freely.

8. Do not forget to *remove* cotton wool after each administration, or you will lose a large amount of Kelene by leakage or evaporation.

9. A No. 70 bottle of Kelene ought to do for six or eight administrations.

10. Don't rush your ethyl chloride at the start; place only about 15 drops on the mask or towel and let the patient get accustomed to the smell; rushing the drug will only make your patient gasp and have a somewhat catchy sensation in the throat.

11. After about a quarter of a minute drop more ethyl chloride on mask, increasing the size of the moistened area to about two inches in diameter, and then in another 15 seconds or so, to an area about the size of palm of hand.

12. The secret of good anæsthesia, with free relaxation, is that of ethyl chloride vapour with free air mixture; if the drug is too concentrated on patient, rigidity may arise.

13. Rigidity in a patient does not mean, as a rule, that the patient is not anæsthetised, but the drug, whether nitrous oxide, ethyl chloride or ether, has been too concentrated on patient without free admixture with air.

14. Surgeons, when operating, should remember that all anæsthetics are poisons, and therefore continue steadily at work so as to get the patient off the operating table as early as possible, without, however, sacrificing skill for speed.

15. Don't tell yarns to students during operation, even if they do bear on the point; a yarn can generally stand over with benefit to patient.

16. Don't try and remove an abdominal tumour as big as your head through a four-inch incision; it only means bruising the walls of the incision and the necessity for anæsthetist to keep patient for an extra quarter or half hour under a poisonous vapour which cannot possibly be of benefit and certainly increases the likelihood of post-operative vomiting and tension on the wounded area.

17. In all cases over 12 years of age where open ethyl chloride is followed by open ether, the writer uses as a routine $\frac{1}{8}$-gr. morphia and $\frac{1}{150}$ atropine, given from three-quarters to one hour before operation.

18. $\frac{1}{8}$-gr. morphia and $\frac{1}{150}$ atropin acts as well as the $\frac{1}{4}$ or $\frac{1}{6}$-gr. morphia $\frac{1}{100}$ or $\frac{1}{120}$ gr. atrophine generally given, and also has the advantage that the reaction of iris is not entirely masked by the morphia.

19. The reason for giving the hypodermic from three-quarters to one hour before operation is to allow time for the morphia to have passed over its stimulating stage and reached the sedative period.

20. The morphia places patient on table in a calmer frame of mind than if none had been given, and the atropine, by inhibiting the vagus, acts beneficially, also lessens the secretion in air passages, and diminishes the likelihood of profuse perspiration.

21. The morphia and atropine, as above, are given in all cases, the presence of albumen in any quantity in the urine making no difference to counter-ordering it.

22. The only disadvantage of preliminary of $\frac{1}{8}$-gr. morphia and $\frac{1}{150}$ atropine is that in some cases the patient passes into a quiet morphia sleep, and when the surgeon makes his skin incision there may be movement of the patient.

23. In using open ethyl chloride for minor operations, always remember to tell your patient to empty his bladder before getting on to the operating table.

24. The free administration of air with an anæsthetic is quite as essential for the welfare of the patient during the stage of anæsthesia as it is for the efficient running of your motor.

25. To put a patient to profound anæsthesia with dilated immobile pupil, loss of corneal reflex and complexion of a purplish white hue is not good anæsthesia.

26. The primary duty of the anæsthetist is towards his patient; by carrying out that duty efficiently he fulfils his duty to the surgeon by maintaining the patient in the best condition during operation and on completion of it, so that the patient is placed in the most favourable condition for rapid return to health.

27. Rigidity in a patient under an anæsthetic does not of necessity mean that he is not fully anæsthetised; it may, and very often does mean, that he is asphyxiated from concentration of the anæsthetic, such as ether vapour, nitrous oxide, ethyl chloride, or any other vapour not allowing free-air supply to lungs.

III.

SECTION OF OBSTETRICS AND GYNÆCOLOGY.

PRESIDENTIAL ADDRESS IN SECTION OF GYNÆCOLOGY AND OBSTETRICS.

By G. ROTHWELL ADAM, M.D., C.M. (Melb.).

BEFORE inflicting on you the time-honoured address from the Chair, permit me to offer my warmest thanks for the honour done in selecting me to fill this honoured position.

Gratification at the fulfilment of one of life's ambitions is, however, tempered by the realisation that an exalted position carries with it certain obligations, of which, and by no means the least, is the choosing of a subject on which to address you that may be not only of interest but of some small value.

In casting about for such a theme it occurred to me that, instead of wading through current literature on recent obstetric and gynæcological studies—be it either diagnostic or therapeutic, and with which I doubt not you are as familiar as I—our time might be better spent in taking stock of our position as regards the other departments of our profession.

To this end let us consider the present position of obstetrics and gynæcology from—

1. Education.
2. The relation between the two subjects.
3. The relation of the subjects to the practitioner.

It may be, because parturition was so common an event, that the framers of medical curricula looked upon it as a physiological process, and therefore not within the fighting line of medical practice; but the undisputable fact remains that obstetrics, and to some extent gynæcology too, has occupied the unenviable position of a Cinderella in the medical course.

We must, of course, recognise that subjects like medicine and surgery form the foundation on which a scientific knowledge of obstetrics and gynæcology must rest, and therefore occupy a position of foremost importance. But surely a branch of medical study like obstetrics, dealing, as it undoubtedly does, with events not only of individual interest, but of national welfare, is worthy of more consideration at the hands of medical educational authorities than it now receives.

The time-honoured certificate of having attended ten or twenty cases under a medical practitioner might, with advantage both to the science of obstetrics and the public, be abolished in favour of a three months' attendance in a properly-equipped maternity hospital. And here the question may be raised as to how far the maternity hospitals under present arrangements are capable of fulfilling their functions as educational centres. It may be admitted that they are, for the most part, officered by experts, who devote considerable time to instructing the students in the diagnosis and management of labour in a scientific manner; and even some institutions maintain an extern department, in which senior students may gain a confidence and a sense of responsibility obtainable only by a personal charge of cases.

But, it may well be asked, does a maternity hospital, however well it may organise these duties, thus fulfil its functions completely? It seems to me the time has now arrived when, to every maternity hospital claiming to be an educational institution, a ward for the study and treatment of ante-natal conditions should be attached. To illustrate my meaning, allow me to mention one group of pathological conditions—the toxæmias of gestation—the study of which, under hospital conditions, would yield a rich harvest to scientific obstetrics, and a national benefit to the public by reducing fœtal and maternal mortality.

The obstacle in the way of establishing such departments appears to be the fear of expense on the part of the management of these institutions, more especially as no apparent practical outcome can be assured until after a prolonged period.

But if the profession would individually point out to the public the benefits—one might say the economic benefits—that would, in time, accrue to the community as a whole, I feel sure funds would readily come forth.

But if this matter be of importance to the scientific study of midwifery, of no less value in its practical aspect is a careful and accurate knowledge of the mechanism of labour.

It may, perhaps, savour of the commonplace to discuss so elementary a subject before an audience such as this; but a fairly long experience has convinced me that the difficulties of labour most frequently encountered by practitioners are due to a failure in making an accurate diagnosis of the position of the presenting part. Some years ago I was requested by a practitioner many years my senior to come and do craniotomy, as he and another practitioner had been endeavouring for two days to deliver a woman of her second pregnancy. On examination the head was found to be presenting, but markedly extended, and the occiput posterior. As a matter of fact, engagement at the brim had not taken place. Rectification of the mal-position was readily effected, and delivery speedily accomplished.

Time will not permit us to enter on a discussion of the merits of Cæsarean section and its alternatives.

It must not, however, be forgotten, whatever our predilection may be, that we have two lives in our charge, and if the march of obstetrical science has put it in our power to save those two lives, fœtal destruction should not be resorted to without grave consideration.

The practice of obstetrics has now, I submit, arrived at a stage when every primipara engaging a medical man to attend her during parturition should be examined regarding her pelvic diameters, as well as the excretory functions; for, although we in Australia have been hitherto singularly free from gross pelvic deformities, the increasing population of our cities and development of factory life in young girls will surely increase the number of such abnormalities.

It is doubtless quite true that factory legislation in this favoured country has improved the conditions of life for the factory labourer, and thus robbed it of many evils; but what legislation is powerless to do is to alter the malleability of the adolescent pelvis. Short hours of labour, good food and housing are of vast importance in building up a constitutional resistance to disease or deformity; nevertheless, it is manifestly impossible to entirely abolish the effects of continuous work in buildings on growing girls.

Overwhelming evidence is available to show that Cæsarean section is a safe procedure, both for the mother and child, when undertaken before futile efforts at delivery have been resorted to. Our efforts in the future should be in the direction of being forearmed by a knowledge of the condition of the pelvis and an appreciation of the causes of delay.

It must be confessed that the difficulties of giving adequate instruction in gynæcology to students are very great; in fact, so surrounded is the practical teaching of gynæcology with difficulties from many causes—in this country, at all events—that it is doubtful if it is wise to attempt more than that of an elementary type.

But, recognising the different attitude assumed by the gynæcological patient towards the unqualified and the qualified man—how they obtain their knowledge is marvellous—I would suggest the institution of a system of clinical assistanceships at our gynæcological hospitals such as has been established elsewhere, notably in London, with beneficial effect, whereby the young qualified practitioner might be able, for a small fee, to see and assist in the work of the hospital.

It is not so much a knowledge of operative work the young practitioner requires as skill in gynæcological diagnosis, and a sound understanding of the natural course of disease.

It is a matter for regret that the fascinations of the operating theatre has a tendency to abolish that patient observation so necessary to attaining proficiency in diagnostic art. The temptation to do something instead of learning everything possible about a case appears to be irresistible.

Fashion in operative procedure is subject to change; diagnostic skill is built on a sure foundation of accurate observation, patient inquiry and logical deduction, and is eternal.

II.—Relation between Obstetrics and Gynæcology.

It might be assumed, with good reason, that these relations were so well recognised that a reference were unnecessary; but it seems to me that attention cannot be too often directed to the undoubted fact that much gynæcology results from bad midwifery.

I would like to see " writ in large letters " on the wall of every obstetric chamber, " Obstetric morbidity leads to gynæcological treatment." If this lesson taught by the gynæcologist was fully appreciated by practitioners, I fancy the hasty use of forceps would be curtailed, the aseptic ritual of the lying-in chamber would be less perfunctory, and details of the puerperium more strictly enforced.

Broadly speaking, more danger ensues from hasty interference with labour than from delay—to the mother. Delay, while not without maternal risk, is more inimical to the child. Obviously, the remedy is to make an early and accurate diagnosis of the cause of delay.

It is a matter of some comment that while the outbreaks of virulent puerperal sepsis have been banished from the community, the gynæcological treatment of women has increased. Of course it is plain that there are many explanations for this condition of affairs, but one factor obtrudes itself, viz., the non-recognition of the mild types of puerperal sepsis. The patient, in all probability, has had a more or less tedious labour, but no serious difficulties have arisen; yet, on the third day, there is a slight rise of temperature, and the lochia becomes offensive. These symptoms usually rapidly subside, and the convalescence, to all appearances, is normal in character.

In many such cases, however, if an examination be made after completion of the puerperium, involution of the pelvic organs will be found to have become arrested, with its train of morbid consequences. It is to be doubted whether the majority of practitioners have yet grasped the importance of the sequential progress of infective processes of a mild character resulting from parturition. Lacerations of the cervix and the vaginal wall open up avenues of infection which lead to arrest of that involutionary process on which depends the restoration of the pelvic organs to a normal state after the complex changes associated with gestation.

Thus we find a heavy uterus, with relaxed ligaments, ready to lose the poise on any provocation, and become displaced, most usually in a backward direction. Nor does the morbidity stop here, for, owing to interference with the blood and lymph circulation consequent on torsion of the broad ligament, chronic changes of a more or less permanent character ensue in the uterine body and endometrium.

It is, however, when we consider the effects of a severe puerperal infection that the obstetrician and the gynæcologist come into closer touch.

The surgical treatment of such infections has for long been the aspiration of the gynæcologist. But can we, at the present day, formulate any definite plan of procedure? I fear the answer can only be in the negative.

At first glance it would appear to be a most rational mode of treatment, removing the prime infective focus, the uterus; but, apart from the well-recognised difficulties of determining the proper time for operation, more accurate knowledge of acute septic processes, and their effects on blood and tissues generally, demonstrate the futility of attempting to stay the infective process by such efforts.

The position is no doubt different where the sepsis is of a more chronic nature, for the patient has time to elaborate her own antitoxin, and if the septic focus be now removed a good recovery may be reasonably anticipated.

On several occasions I have removed the uterus by supra-vaginal hysterectomy, with most satisfactory results, where the disease had assumed a chronic course, with exacerbations at intervals. On making sections of the uterus, subsequent to removal, numerous abscesses were found scattered through the organ.

In another type of puerperal infection, especially of a phlebitic nature, excision of the pampiniform plexus, or removal of one or both ovarian veins, as high as may be necessary, has been advocated.

These cases seem to me to be infrequent, and a note of warning against indiscriminate exploratory operating, in the hope of finding thrombosed veins, may be sounded.

There can be no doubt concerning the usefulness of the operation in suitable cases—the difficulty lies in selecting the right case; and it must not be forgotten that these cases stand operation badly, so that if a wrong diagnosis has been made the chances of recovery have been seriously jeopardised.

So far as experience indicates, surgery does not at present, except in a few favoured cases, lead to a short cut in the treatment of puerperal sepsis of an acute character.

III.—The Relation of the Subjects to the Practitioner.

Does the study of obstetrics and gynæcology help the practitioners to a better understanding of medicine and surgery? The answer to this must be, I take it, in the affirmative. Numerous examples must be apparent, on reflection, where a practical acquaintance with these subjects has thrown a clearer light on a doubtful medical or surgical problem.

But while this practical benefit must not be ignored, it is in the domain of physiological and pathological science that the simpler, probably elemental, processes are revealed, shedding a new light on obscurities hidden by the complexities of a medical case. For example, when Lawson Tait demonstrated the spreading of an infective endometritis to the Fallopian tubes, and thus leading to a pelvic peritonitis, professional opinion was prepared to understand the development of a general peritonitis from an infective condition of the vermiform appendix.

Considering the trend of modern physiological research, few fields offer a more bounteous reward than those of obstetrics and gynæcology. To take one illustration: the function of the ovary, with its more than probable dual function of ovum-bearing and some internal secretion—and indications are not wanting that shortly a third role will be assigned it of regulating the periodicity of menstruation by means of a lutin secretion.

I should like to see established, in connection with our obstetric schools, a research scholarship for the express purpose of encouraging investigations in obstetric physiology and pathology. I feel certain it would be by some such means as indicated that further progress in obstetric knowledge will be made.

Technical skill has, for the present at all events, far outstripped our physiological and pathological knowledge; and, indeed, it is to be feared, in gynæcological practice, led to a *cacoëthes operandi* not always to the benefit of the patient.

The ability to do does not necessarily mean the highest skill; rather is it the capacity to understand. Technique should, in all cases, be subservient to knowledge.

The daily routine of practice has a tendency to confine attention to one side of professional duty—an all-important one, no doubt—of caring for the immediate wants of patients under our care. Nevertheless, I take it that there is an obligation incumbent on each one of us to add something to the sum-total of current medical knowledge. No observation, if accurately recorded, is without its value. A Jenner noted the similarity of cowpox to smallpox, and a marvellous superstructure of immunisation has, indirectly it may be, resulted therefrom.

Some interesting problems, all of a speculative nature, are raised regarding the apparent connection between reflex nerve disturbances and uterine displacements, more especially when there is cervical laceration, with ectropion and chronic cervicitis. A case exemplifying this came under notice some years ago, and may be briefly cited. A nulliparous woman developed a persistent cough, with loss of flesh; so much so that she was sent to a distant State, as she was believed to be suffering from early phthisis. After eighteen months' sojourn there, without gaining any weight, she returned to Victoria and consulted a physician, who could find no evidence of local disease. Her larynx was also examined with a negative result. As her history disclosed some menstrual abnormality, she was sent to me for a gynæcological examination, with the result that a marked retroflexion of the uterus was found, with endometritis. The patient declined radical treatment for the displacement; so, after curettage, a pessary of the Hodge type was fitted, which kept the uterus in good position. Within a few months her cough disappeared, and she increased in weight. How much blame may be attached to the uterine displacement, or how much depended on the nerve element in such a case, it is hard to determine; but it is curious that if she went without her pessary for any length of time, and the uterine displacement recurred, the cough returned.

It is, however, in the relation between pelvic disorders and the so-called reflex nervous phenomena that extreme caution has to be observed. The days are passed when important organs, such as ovaries, are credited with being the source of diverse ills, from an insanity to a pain in the left iliac fossa, and therefore to be promptly ablated.

As pathological experience has advanced, so the removal of these organs for hypothetical disease has decreased.

It may be laid down as a sound rule of practice that these organs should never be removed for the mere relief of iliac pain unless indisputable evidence of disorganisation be forthcoming.

The fortitude with which a woman will face the terrors of the operating theatre compels our admiration, while the nerve demoralisation brought about by some apparently trivial pain is almost past comprehension. Such temperamental contradictions demand very careful consideration, and tax the judgment of the gynæcologist to the utmost.

The only true and safe guide, under such circumstances, are a dispassionate inquiry into the case, and a disregarding of all hypothetical views.

The vexed question of the connection between insanity and gynæcological disease cannot be settled merely by the report of successful operative procedures. As experience is gained the impression grows on one that mental improvement is not so much due to the pelvic treatment as to the improvement in the general health that may follow such treatment. Indeed, cases are not uncommon where operative treatment is followed by an increase in the mental aberration, and benefit is not obtained until a lengthy period has elapsed.

It is inadvisable to undertake gynæcological treatment in the insane unless the evidence is plain that the pelvic disorder is directly causing health deterioration.

The conclusion of the whole matter is: that in more accurate knowledge regarding physiological and pathological processes of the female pelvic organs lies the way towards advance in obstetric and gynæcologic progress.

It may be that the future will show the labours of the present generation in overcoming technical difficulties in operative procedures, whilst not altogether in vain, do not lead to the ideal treatment of disease. Already the clinical pathologist is showing us that the natural resources of the organism can be utilised by means of vaccines to combat infective processes.

Gonorrhœa, that scourge of the human female, appears to be amenable to vaccine treatment; and although puerperal sepsis has hitherto proved too complicated an infection to respond satisfactorily to such methods of treatment, the time cannot be far distant when the difficulties will be overcome.

The study of obstetrics is of such individual and national importance as to entitle it to rank with medicine and surgery on equal terms in a medical curriculum; and it can only do so by being afforded similar facilities for instruction.

I am well aware of my inability to convey to you all I would wish to express regarding the subjects touch upon in this address. But if I have been able, in ever so small a way, to suggest lines of thought, or stimulate any to further study of so worthy a speciality, I shall feel my endeavour has not been in vain.

AN INTRODUCTION TO A DISCUSSION ON THE ETIOLOGY OF ECTOPIC GESTATION.

By Felix Meyer, M.D., B.S.

Honorary Gynæcological Surgeon, Women's Hospital, Melbourne.

An inquiry into the causation of ectopic pregnancy is beset with difficulties which relate, for the main part, to the interpretation of evidence.

At the outset there is a difference of opinion regarding the histology of the normal Fallopian tube, most authorities agreeing that the lining epithelium is made up of more than one layer of cells, while Schridder, as the result of a searching histological investigation on the purulent inflammations of the Fallopian tubes, affirms that the epithelial lining is made up of a single layer of cells, which he differentiates into ciliated and secretory.

Again, a variety of pathological conditions, not necessarily related, associated with pregnant tubes, are accepted by various observers as etiological factors. Such, *e.g.*, are appendicitis, adhesions of tubes to the peritoneum, &c., myomata, tubal polypi, fœtal and atrophic conditions of tubes, &c. In addition, the problem is confused by the practical impossibility of studying the relation of the fertilised ovum to the tube at the initial stage of implantation; the original conditions determining the implantation being merged into, if not completely changed or lost by the rapid topical and structural alterations of development; and thus important evidence is wanting in the exact determination of the etiological factor.

A very real difficulty also arises in making a definite pronouncement on the causal connection between observed conditions—mainly histological—of the pregnant tube, some regarding as the cause what others assert to be the effect of the arrested ovum. Among the latter is such a careful investigator as Couvelaire.

And Bland Sutton, for example, is of opinion that the occlusion of the fimbriated extremity of the pregnant tube, so very often noticed, is an effect of the pregnancy.

The same difficulty of deciding what is cause and what is effect has been raised by Dr. Constance Ellis when going over sections of pregnant tubes with me in the Bacteriological Laboratory under her charge in the Women's Hospital, Melbourne.

An inquiry into the reasons why the fertilised ovum should be arrested and continue to develop at some point in its passage along the Fallopian tube to the uterus must obviously deal with—(*a*) Conditions of the tube offering mechanical obstruction to its passage; and (*b*) conditions which favour its development.

Whether in some cases the ovum itself may at a very early stage carry with it conditions which favour its arrest and development in a perfectly normal tube, or whether a perfectly normal ovum may develop in a perfectly normal tube, are questions which also enter into the solution of the general problem. Bland Sutton, in 1896, made the statement that, "as a matter of fact, the evidence now indicates that a healthy Fallopian tube is more likely to become gravid than one which has been inflamed." Such a dictum was based on the circumstance that he had met with cases of tubal gestation without any evidence of existing inflammation; and, in addition, it was at that time (1896) the generally accepted belief that conception took place in the uterus. To-day the bulk of evidence points to the outer part of the tube as the most common site of the meeting of the spermatozoon with the ovule, though this may occur at any point between the surface of the ovary and the uterus itself.

Of forty-two cases of ectopic gestation for which I have operated, thirty-four were multiparæ, eight nulliparæ. They included one case of bilateral tubal pregnancy, one of simultaneous tubal and uterine pregnancy, one of tubo-uterine, and one which I originally regarded as a primary abdominal pregnancy, but which I am inclined to regard now as a case of very early tubal abortion which developed abdominally.

Among the multiparous cases, the shortest period between the last labour and the ectopic gestation was two years; the longest nine years. In more than one-third of all the cases there was a definite history of gonorrhœa. The influence of puerperal sepsis cannot be fairly stated; in some of the cases the history clearly pointed to an infection of the puerperium. Where the condition was diagnosed or met with before rupture, deliberate examination of the opposite tube was possible; and removal of that tube or some conservative measure was carried out in fourteen cases. But, with a full appreciation of the limitations of macroscopical evidence, I am not prepared to say that these fourteen cases represented the total abnormality of the non-pregnant tubes, though, so far, I have not met with a case of repeated ectopic gestation amongst my cases—of which a third were "urgent"—calling for immediate and rapid operating in hæmorrhaging or exsanguined woman, allowing no time for any secondary procedures.

The conditions noted at the time of operating were mostly inflammatory—salpingitis of the catarrhal type, sactosalpinx, adhesions of the tubes to peritoneum, uterus, or to Douglas's pouch. Pus was present only in one case, a small tubo-ovarian abscess of a non-pregnant appendage. Uterine myomata were twice met with, neither tumour being large; cystic ovaries were very common, but no ovarian cyst was present, and there was no evidence of tubercle.

Curiously enough, since this paper was begun a few weeks back, I removed within ten days of one another two pregnant tubes, unruptured. Both were in married women, one married sixteen years, the other five, neither previously pregnant.

Dr. Cuscaden had done a right salpingo-oophorectomy on the latter twelve months previously; while I had removed, in the former, seventeen months before, an ovarian abscess and pysoalpinx, leaving the other ovary and tube, which were free of adhesions, and apparently little affected.

In another case, one of those early ones in which I began conservative work, a young married woman not previously pregnant, I removed a large pus tube from one side; the opposite tube, in a less degree pathological, contained a thin mucous fluid, which I evacuated with an inch-long incision along the upper surface, closing it with a fine suture, leaving, as I believed, a clear lumen. Four months later this tube was removed by a surgeon in the country for an ectopic pregnancy.

Still more convincing was the result of making an artificial abdominal ostium in a sactosalpinx and fixing the new fimbrial part to the ovary, the other appendage demanding removal; a few weeks later she returned to the hospital, when I cut down on the pregnant tube. Three of these cases gave a clear history of gonorrhœa, the fourth a doubtful one.

In cases such as these it would be difficult to escape the conclusion that the conservative measures adopted were mainly, if not wholly, responsible for the tubal pregnancies which followed them—an experience, so to speak, of tubal pregnancy in the making.

In the forty-four cases of this series, two points may be emphasised—(1) That salpingitis, gonorrhœal or septic in origin, was the most frequent lesion, and in most cases bilateral; (2) that a varying period of sterility, usually less than two years, existed between the last labour and the tubal pregnancy.

Franz found inflammatory changes in the tubes in 80 per cent. of those cases where there was a sterile period of two to seventeen years, and in 53 per cent. where the sterile period was less than two years.

In an analysis of 214 cases of ectopic pregnancy at the Massachusetts General Hospital, W. P. Cones records cystic ovaries, disease of the opposite tube, adhesions or a history of a previous miscarriage, in over 83 per cent. of 202 cases.

Mandl, Schmidt, and Dührssen found a history of gonorrhœa in 66 to 68 per cent. of their cases.

Ahlfield, on the contrary, saw only one case of ectopic pregnancy in twelve years' experience in Marburg and Giessen, a result due, he believes, to the rare occurrence of gonorrhœa in the surrounding country.

In marked contrast is Ladinski's experience of 150 cases of tubal pregnancy operated on by him in sixteen years, mainly in the Beth Israel Hospital in New York. Rabinowitz, commenting on this, says:—" I ascribe the enormity of this material in his service to the prevalence of gonorrhœa in the immigrant women. The husbands of these women, who have sojourned in this country for several years before the arrival of their wives, have during this period of enforced celibacy contracted gonorrhœa."

Finsterer, on the other hand, discounts the influence of gonorrhœa as a predisposing cause of ectopic pregnancy.

In his series of 133 cases, gross affections of the appendages were present in twenty-eight cases only, milder changes in eight. Of 120 women, ninety-seven had a history of a normal puerperium previous to the ectopic pregnancy. He assumes that a woman is hardly likely to be infected by gonorrhœa for the first time, after she has had several children, an assumption negatived by my own experience.

Bearing in mind the fact that prolonged periods of sterility, associated with a history of gonorrhœa or puerperal sepsis in women who have not been operated on, are often followed by normal pregnancy, there is justification for the conclusion that in those cases where a period of sterility was followed by an ectopic pregnancy the tube had so far progressed towards the normal condition as to allow of the passage of the ovum along some portion of it. It is eminently reasonable to suppose that in the case of a tube which has long ceased to functionate, and is reacting under the stimulus of the physiological factor, there must be some period before it again attains a normal state at which the conditions are favourable to the implantation of an ovum.

The view that the inflammatory conditions of the tubes, found so largely in tubal pregnancy, explain the mechanical obstruction to the progress of the ovum, is strongly supported by histological evidence.

I may here call to mind that the Fallopian tube is made up of three layers—an inner or mucous layer, with an epithelial lining of a single layer of ciliated and secretory cells; a middle or muscular layer, made up of an inner circular and an outer longitudinal layer; and an external or peritoneal coat, beneath which is a layer of connective tissue of varying thickness, the subserous coat.

J. Whitridge Williams, in connection with the normal anatomy of the tube, calls attention to an abnormality in its development which he believes may bear a causal relation to the production of extra-uterine pregnancy, viz., diverticula extending from the lumen into the wall of the tube, reaching almost to its peritoneal covering. They are lined by the typical single layer of ciliated epithelium, and correspond in all respects to the structure of the normal lumen of the tube. They should not be confounded with the so-called accessory ostia of the tube.

Inflammatory processes of the tubes due to infection differ in degree and effect. In the mildest forms, with hardly any symptoms, the ciliated epithelium may be affected, and function partly arrested. Such is a very common result of mild puerperal sepsis.

With a greater degree of infection the mucosa is more deeply affected, and swelling and exudation result in the fusion or coalescing of the adjacent folds. This condition has been carefully investigated by C. D. Williams, who points out that the fusion may take place between the folds projecting from the opposite end of the lumen, as well as between adjacent ones. The enclosed epithelium is preserved, and the cilia are usually present, at least in part. These little canals may be patent at one or both extremities, but more often are blind at the mesial, and open at the distal end.

These crypts or culs-de-sac, with the resulting cribriform structure, are to be seen in most cases of follicular salpingitis. I have seen them in sections of my cases made by Dr. Ellis, both in the pregnant and non-pregnant tube. The muscle-layer in some sections was thinned and flattened, in others not markedly affected. Sometimes it was replaced by a fibrinous exudate invading the peritoneal coat, and extending inwards.

These "false diverticula," as C. D. Williams calls these epithelial-lined canaliculi, were found by him both in the pregnant tube and the opposite one, where both tubes were removed, in all cases. He considers them the chief predisposing cause of tubal pregnancy in 95 to 98 per cent. of cases.

Opitz, in 1902, found the canaliculi resulting from the inflammatory adhesion of the neighbouring longitudinal folds in every one of twenty-three pregnant tubes examined in serial section. He, too, held that their existence afforded a possible explanation of the occurrence of ectopic pregnancy.

It is easy to understand that with the distal extremity opening fully to the lumen of the tube the ovum could enter and become arrested at the blind end.

The same net-like or honeycomb appearance of the lumen on the uterine side of the ovum resulting from the fusion of the villous processes of the mucosa across the lumen was found by Drs. Wade and Watson in their examination of an early tubal gestation of two to three weeks' duration.

In addition to C. D. Williams and Opitz, Bandler, Gitch, Schauta, Dührssen, and others regard inflammatory affections of the tubes as the commonest cause of ectopic gestation.

Peritoneal adhesions of the tube, binding it down and causing kinks of the lumen, have been regarded as a cause of tubal gestation. With a tube anchored by firm adhesions, diminished ciliary current and peristalsis checked by marked loss of muscle, the possibility must be admitted.

External Migration of the Ovum.

From the circumstance that the true corpus luteum is often found in the ovary of the opposite side to the pregnant tube, external migration of the ovum has been advanced as an explanation of ectopic pregnancy, the theory being that during its transpelvic journey the ovum became enlarged by a growth of the trophoblast to an extent which prevented its progress in the tube. Against this must be put the simultaneous occurrence of uterine and tubal pregnancy, and the much greater proportion of cases where the true corpus lutem is found in the ovary on the same side as the pregnancy.

Peters and v. Spee further assert that chorionic villi do not appear till the ovum has been nourished for some time by the decidua developed in the tube.

Vineberg has reported a case of ectopic gestation occurring on the stump of an excised left tube which had been removed with an ovarian abscess, the other adnexa being left intact as normal. A good-sized corpus luteum was found in the right ovary, and Vineberg considered that the period occupied in the transpelvic journey of the ovum accounted for its development and abnormal implantation.

On two occasions on which I removed the ovary of one side and the tube on the other I have seen normal pregnancy follow. Personally, I am disinclined to accept the theory of tubal implantation of the ovum from delayed transit.

Webster's Theory of Decidual Reaction.

From the fact that he has demonstrated the existence of decidual reaction in the tubal mucosa in all his cases of tubal pregnancy, Webster believes that the fertilised ovum can develop only in tissues capable of undergoing a genetic reaction—that is to say, in Mullerian tissue.

This deciduation he regards as a reversion to an earlier type, a retrograde change.

This theory is invalidated by the statement of Bland Sutton that in his extensive work among the animals in the Zoological Gardens in London he has not met with a true case of extra-uterine pregnancy.

Nor are authorities agreed as to the finding of true decidua—decidua of maternal origin—as a development from the tubal mucosa, some asserting that there is little, if any; others, that the placenta is entirely fœtal in its origin. That this latter condition does occur is, I think, demonstrated in the following case, which also proves that, contrary to Webster's view, primary development may be established on a peritoneal surface:—In July, 1904, I removed from the abdomen of a woman, who had had her first and only labour more than two years previously, a fœtus between the fourth and fifth month, with the fœtal head attached to the omentum by strong and highly-vascularised adhesions. The pregnancy dated back twelve months, and the fœtus was undergoing a change of coagulation necrosis. The placenta—a firm, hard mass, with a greatest circumference of 12 inches—lay in the mid-line attached to the upper surface of a small uterus. Both tubes and ovaries were quite free, the right tube lying with its fimbriæ resting on the right upper lateral surface of the fundus. Both tubes were apparently in every respect normal; an ordinary probe was passed freely along each tube. The ovaries were cystic. An uneventful recovery was followed fourteen months after by labour at full term.

If this was a case of primary abdominal pregnancy—that is, if the fertilised ovum never at any time entered the tube, but found its way immediately on to the uterine peritoneum—its subsequent parasitic development is capable of explanation independently of the necessity of Mullerian tissue. I was careful to dissect off a thin layer of the subplacental uterine tissue for histological examination, and the sections made by Dr. Ellis at the time satisfied me that the decidua was of trophoblastic origin. And if the case be considered one of very early tubal abortion, the line of adhesion between the fibrous capsule of the placenta and the uterine muscle, which had become thoroughly organised, still supported this contention.

I have endeavoured to compress this introduction to a discussion on the etiology of ectopic gestation within the limits of the time prescribed, and have left to others the discussion of etiological aspects which may have been suggested by, or which have come within, their experience, but not mine. Thus, I have not dealt with infantile or fœtal conditions or puerperal atrophy of tubes, or appendicitis, as causal elements; nor have I had the good fortune to meet with a case of ovarian pregnancy.

But as a summary of my experience and study, I believe that in pre-existing inflammatory conditions of the tubes, chiefly gonorrhœal in origin, is to be found the main, though not the only, predisposing cause of tubal pregnancy.

And as a corollary, from personal experience, I am strongly of opinion that so-called conservative surgery of the adnexa—whether it takes the form of operative measures for the correction of abnormal conditions, or the leaving of those conditions *in statu quo*—must be based upon a serious consideration, in every case, of the possible advantages, from the prophylactic standpoint, of radical procedures.

SYMPTOMS AND DIAGNOSIS OF ECTOPIC GESTATION.

By J. A. G. Hamilton, B.A., M.B. (Dub.), L.R.C.S.

I think I am safe in saying there is no condition in surgery where a prompt and accurate diagnosis is of more importance than in ectopic gestation, but unfortunately the diagnosis in some cases is by no means easy, and it is generally the general practitioner and not the expert gynæcologist who sees these cases first; but even to the expert it is often impossible to distinguish absolutely between some cases of ectopic gestation and some cases of pelvic infection without an exploratory incision.

The study of these cases as they come to the hospital have shown me that they are occasionally most difficult of diagnosis, but in the majority of cases comparatively easy by a careful examination of the subjective symptoms. In the case of the general practicioner, physical examination may often lead him much further astray, than if he relied on the subjective symptoms. I often have cases of tubal pregnancy sent to me by the general practitioner in which his description of the subjective symptoms is correct, but he has got astray when he came to describe the objective symptoms. It must be always remembered, however, that tubal pregnancy may occur without any symptoms. A woman apparently in perfect health, thoughtless of any danger, may die in a few hours from acute hæmorrhage—the first symptom of her condition.

The symptoms of an ectopic gestation are depending on the direction and course of the pregnancy, but they are not characteristic; therefore, the patient is usually entirely unaware of its existence until rupture or tubal abortion occurs. In many instances the patient considers herself normally pregnant. She may have slight pain in one or other ovarian region to which she pays but little attention; usually, however, the early occurrences of some of the accidents of tubal gestation attracts her attention. Before such accident or complications arise, there are most frequently no subjective symptoms to excite any suspicion of this peculiar form of pregnancy. The first indication of the abnormal condition is a severe lancinating pain in the lower abdomen, often described as " a feeling of something having given way." In severe cases a woman becomes semi-conscious, is covered with a damp perspiration, has sighing respirations, and presents all the symptoms of most profound shock—complains of intense abdominal pain if conscious enough, and a frequent, often scarcely perceptible, pulse. Sometimes the symptoms are not so marked. The patient is weak and shows signs of loss of blood, but soon rallies, with recurrent attacks of similar character, which indicate that the hæmorrhage has again recurred. A good deal depends upon whether the gestation sac has ruptured into the peritoneal cavity or into the broad ligament. In the latter, the symptoms are not nearly so marked—the indications of collapse are slighter, as the resistance of tissue limits the amount of bleeding; but when the rupture takes place into the peritoneal cavity, there is nothing to arrest the hæmorrhage and fatal symptoms if intervention is not instituted early. A bimanual examination in the broad ligament rupture will reveal a tense and tender mass bulging into the vagina and pushing the uterus to one side; the mass may extend behind the uterus and surround the rectum.

Bimanual examination after an intra-peritoneal rupture reveals an indefinite fulness in the pelvis behind the uterus. The blood free in the peritoneal cavity coagulates slowly, and the fluid blood and unrestrained clots are often very difficult to palpate. For this reason the examiner will at first only feel an ill-defined fulness in the pelvis. Fehling points out that in his belief there is a substance present in tubal rupture which delays coagulation, which accounts for the fact that a small tubal opening will bleed so profusely, while in a uterine rupture the hæmorrhage is often moderate, although the vessels are much larger. If the woman survives the hæmorrhage, and the mass of blood becomes solid, it may be distinctly palpated as a solid mass behind the uterus, bulging into the vagina and pushing the uterus forward, causing urinary tenesmus—a constant symptom due to the sudden interference with the capacity of the bladder, consequent upon the development of the hæmatocele.

It is not often an opportunity occurs to diagnose tubal pregnancy before rupture takes place. I think most of the cases are discovered or operated upon by accident, the operation being performed under the diagnosis of pyosalpinx, or some other disease of the tube. These cases show the value of the general rule to operate without delay for all gross diseases of the tubes. If it should turn out to be an ectopic gestation, the woman is saved the imminent dangers associated with a developing tubal pregnancy. If the physician should be consulted before the death of the fœtus, the diagnosis may be made with a near approach to accuracy if menstruation has been absent for one or two periods, if the uterus is soft and somewhat enlarged and elongated, if there is subjective and objective signs of pregnancy, and if there is a soft elastic and tender tumour involving one tube. Voit points out that the soft elasticity of the tumour is almost pathognomonic.

The special symptoms pointing to tubal pregnancy after rupture (but not pathognomonic of it) are as follow:—

I. A missed period.—The patient previously regular fails to become unwell at the proper time—she goes overtime a few days or weeks; the suppression of menses does not occur so regularly in extra-uterus as in normal pregnancy.

II. Sudden onset of pain.—After going overtime for a few days or weeks, the patient is suddenly seized with pain in the lower abdomen. The pain is usually severe enough to confine her to bed for a day or so—in some cases it is accompanied by all the symptoms of profound anæmia.

III. Blood-stained vaginal discharge.—Usually about the time of the onset of pain a blood-stained vaginal discharge appears. The patient often regards this as a return of her menstrual period, but usually it is not as free as her usual period, and does not stop in a few days as the menstrual flow should, but persists as an irregular bleeding, some days present and other days absent. This, with the pain arising from the rupture, may cause the woman to believe that an abortion is about to occur. This suspicion is still further confirmed by the discharge of a cast of the uterus or shreds associated with clots, which may and often does deceive both the patient and her attendant into the belief that an abortion has occurred.

IV. Only slight fever.—The temperature is rarely high. Noble has reported cases in which the rupture and hæmorrhage has been associated with a rather rapid and marked rise of temperature. The general rule, however, is, that when the hæmorrhage is marked, the temperature is subnormal, as would be expected in cases of shock and sudden collapse. A few days after the hæmorrhage the temperature may be raised, but not high. The rise is due possibly to the fibrous element in the collection, but more likely to some infection by organisms from the intestinal tract.

V. Evidence of internal hæmorrhage.—This will, of course, vary with the amount of blood lost. If the hæmorrhage is free the patient will be in intense collapse in a few minutes after the onset of pain; in other cases the internal bleeding is slight, and produces very little effect on the patient's pulse; but there is generally some pallor, and the presence of the hæmorrhage, however slight, causes pain.

VI. Exacerbations of pain without apparent cause, and without marked rise in temperature.—This is fairly characteristic of tubal pregnancy, in which there are repeated slight internal hæmorrhages. In salpingitis, such excerbations of pain could be caused only by a leakage from the infected tube, and this would be accompanied by a decided rise in temperature and a rapid, full, rather than a small running pulse.

VII. Signs of pregnancy.—Some of the earlier signs of pregnancy are generally present. Stomach disturbances, mammary changes, slight enlargement of the uterus, discoloration of vagina, softening of the cervix.

As regards the cervix, Pinaud points out "that the modifications of the cervix in extra-uterine pregnancy is not properly interpreted. In reports we read 'cervix small, firm, not softened.' These expressions are misleading. The cervix always becomes softer in pregnancy, whether extra or intra-uterine, but it must not be forgotten that the softening process passes away when the ovum dies. The cervix then is firm, for obstetrically speaking there is no pregnancy—nothing but a fœtal sac."

If in addition to these symptoms the woman gives a history of more or less extended sterility, of attacks of pelvic inflammation, especially of gonorrhœal origin, it is a point in favour of ectopic gestation.

I have made an analysis of the symptoms in thirty-four cases of tubal gestation I have seen within the last two years.

(1) A history of amenorrhea was obtained in twenty-three cases.
(2) Acute pain with faintness occurred in twelve; acute pain without faintness in sixteen.
(3) Indefinite and less severe pains in the other six.
(4) Irregular vaginal bleeding occurred in twenty-eight cases.
(5) Signs of pregnancy were noted in twelve cases.
(6) A decidual cast was passed in three. In one case operated on a few days ago, the woman brought into the hospital with her a beautiful cast of the uterus, under the impression it was an abortion.
(7) Sterility extending over six years was noted in thirteen cases; eight of the cases occurred in unmarried girls.

I will read the notes of two cases I operated on. One shows a typical history of ectopic gestation; in the other, the history rather pointed to appendix trouble.

I.—H.F., aged 23, single; admitted on evening of June 5, 1909. General health good; was seized with a sudden severe pain in lower abdomen on morning of admission, with faintness. Pulse almost imperceptible, pallid, with a pinched and anæmic expression; great tenderness in abdomen, especially on right side, and muscles tense. Gave history of irregular bleeding from vagina for about six weeks. Has not passed clots or shreds; fluid in breasts, denies pregnancy.

P.V.—Vagina discoloured and soft cervix softened, shortened and patulous. An elastic fluctuating mass felt in Douglas' pouch, continuous with a similar mass felt above pubes. Operation same evening, abdomen opened in middle line. When peritoneum was opened a large quantity of blood and clot escaped; the right tube was found enlarged and showing signs of a recent rupture; as the ovary was enlarged and adherent it was removed with the tube.

II.—M.C., aged 35, married; admitted October 19, 1910. Complained of pain in right side; has had it for about five years, during which time she has had three definite attacks. Pain comes on quite suddenly, and is generally accompanied by vomiting. Commences on upper part of abdomen and extends all over abdomen, but is worse on right side. The last attack commenced five weeks ago, and has gradually subsided, although pain was rather severe this morning. Pain is always relieved when she passes water. Three children, youngest 2 years. Three miscarriages; one after first and one after second attack, thinks she had one after last attack. Menses regular (twenty-eight days), three to four days until nine weeks ago; thinks she miscarried fourteen days ago; since then has had a blood-stained vaginal discharge, not profuse. Temperature, 99; pulse, 132; respiration, 30. Abdomen tender on right side, some rigidity of muscles on that side.

P.V.—Uterus pushed forward by an elastic mass filling Douglas' pouch, and extending to right of uterus. Douglas' pouch opened, a large quantity of dark tarry blood or clots escaped. Sausage-shaped mass felt in right side of uterus, this proved to be the right tube distended with blood and showing a rupture near the abdominal ostium. The tube was removed, leaving the ovary; there was no evidence of fresh bleeding. The tube on examination shows a gestation sac, containing an embryo 1 cm. in length, in the centre of decidual material.

This was one of those cases in which an exact diagnosis was impossible, her symptoms pointing more to appendicitis, and the opening in the posterior cul-de-sac was really made for diagnostic purposes. I prefer operating for ectopic gestation by the abdominal route. I frequently use the vaginal route for diagnostic purposes in doubtful cases—if it is an old rupture the vaginal drainage is sufficient; if there is fresh bleeding, the ruptured tube can be removed through the vagina, but it is safer to secure the bleeding vessel by opening the abdomen, if the condition of the patient will stand an abdominal operation.

THE TREATMENT OF ECTOPIC GESTATION.

By Arthur J. Nyulasy, M.R.C.S. (Eng.), &c.
Gynæcologist to the Perth Public Hospital.

In a short paper on the treatment of ectopic gestation it will be impossible to do much more than indicate those great basic principles which should alike be the surgeon's guide and the patient's safeguard. Of these, none seems to me to be of such far-reaching importance as—(1) early diagnosis, and (2) prompt operation. If the supreme significance of these two factors were completely realised, the mortality of ectopic gestation would be reduced to perhaps 3 or 4 per cent. or less. We would cease to encounter those cases where operation has become difficult and dangerous from the presence of dense extensive adhesions, and from the existence of marked anæmia due to repeated hæmorrhages. We would no longer meet with those even more formidable instances of ectopic gestation close to term which have been at once the reproach and the despair of gynæcology. Sepsis, too, in an unoperated ectopic gestation would be a nearly unknown surgical disaster. All these would have largely become theoretical considerations in treatment—for practical purposes they would have disappeared in its evolution.

Early Diagnosis.—A grave responsibility rests upon teachers of gynæcology to impress upon their students the life-saving value of an early recognition of ectopic gestation. If there is the slightest suspicion of the condition, the most alert watchfulness is called for to prevent an almost unnecessary catastrophe. But, assuming the practitioner exercises all care, a remarkable want of intelligence is occasionally displayed by the patients. Thus, a woman taken with pain and collapse decided to come to hospital, but presently feeling rather better proceeded to do some preliminary shopping. While so occupied, she collapsed once more, was brought to hospital in a moribund state, and succumbed before operation could be carried out. At autopsy, the abdomen was found positively full of recent blood and clot, while a two months' fœtus in its membranes floated free in the pelvis.

Prompt Operation.—Speaking generally, the treatment of ectopic gestation, except in early broad ligament cases, should be prompt operation. And this, notwithstanding that cases ruptured into the peritoneal cavity are not seldom encountered in which Nature is making effective efforts to aseptically dispose of the gestation. If she could always secure this end without danger to life or without leaving destructive evidences of her efforts, all would be well. Unfortunately, this is not so. Even if death is not rapid from hæmorrhage, infection may occur later. In this latter contingency, if the blood is walled off, vaginal section is commonly effective treatment. If it is not so shut off, general septic peritonitis ensues, as in one of my hospital cases which had been criminally interfered with in the belief that gestation was uterine, and in which, in spite of vaginal and abdominal section, there was a fatal ending. If on cœliotomy we find old blood clot from tubal rupture or abortion, we may with some justification assume that the clot would have ultimately become absorbed. Thus, in a nullipara in the Perth Hospital, with a rather obscure history, and with the uterus about 5 inches in length, the question of ectopic gestation arose from the presence of marked anæmia said to have followed on left iliac pain and vaginal blood discharge some weeks earlier. On curettage, I found the lining of the uterus a thick friable membrane, which proved to be a decidua. As there was no urgency, and the patient required building up, I did not open the abdomen till a fortnight later, the patient being carefully watched in the meantime. On cœliotomy (23/9/1908) I found a firm rounded mass of old blood clot as large as a turkey egg fixed to the fundus uteri. This mass had worked over from a rent in the left tube, and while fixed to the uterus was also adherent to the abdominal wall. The total effect had been to suggest an enlarged uterus, due perhaps to a somewhat soft fundal fibroid. There were many other large blood clots, and numerous small ones were tightly adherent to the intestines and omentum, there were extensive intestinal and omental adhesions, and at the finish, Douglas' pouch was a ragged freely-oozing cavity, which was gauze-packed on the score of urgency. The right tube was sealed up by adhesions. In this case it is possible the patient would have recovered without operation, the clot becoming slowly absorbed. But at what a cost ! Doubtless adhesions have followed on my operation, although so far the patient has not suffered, but there is, I think, no doubt that if the case had been left to Nature, the adhesions would have been far more extensive, probably far more dense, and far more likely to prove a source of ultimate danger. At the same time, while the clot was being absorbed, there would have been the ever-present risk of infection. If, on the other hand, the case had been operated on at the time of rupture, subsequent adhesions would have been few, if any, and the case might well have stood as an illustration of the value of early diagnosis and prompt operation.

For convenience, I have divided ectopic gestation into two stages—(1) The præ-placental stage, roughly corresponding to the first three months ; and (2) the placental stage, to the remaining period of gestation.

(1) The Præ-placental Stage.

My usual practice is to promptly operate except in early broad-ligament cases in which the extravasation is aseptic, and small. My preference is for the abdominal route, and conservative principles are applied to the ovaries and uterus where possible. In early tubal cases the gestation may be removed without sacrificing the tube, but it is questoinable whether it is wise to conserve a tube which has already proved a failure. In selected cases operated on subsequent to rupture, the vaginal route may be adopted; where infection has occurred in a hæmatocele, it is the route of election. The expectant treatment has been applied with success in pelvic hæmatocele, but for reasons already suggested I advise abdominal cœliotomy, and especially if the hæmatocele is large or is causing symptoms.

Operation.—If in the presence of collapse from hæmorrhage we could be sure the bleeding had ceased and would not recur, it would be wise to wait for reaction. Since there is no such guarantee I never hesitate to operate as soon as practicable. In these urgent cases practically no preparation is required further than painting the abdomen with a 2 per cent. iodine tincture. Of anæsthetics "open" ether seems fairly safe, while the Trendelenburg position is usually adopted, and pituitrin may prove useful. An intra-venous saline should in very urgent cases be given immediately active bleeding is controlled. As a matter of fact, I have only occasionally found the ovarian artery spurting, so that there will probably be no necessity to clamp off the offending tube, and this structure can be dealt with as almost the final step. Having then controlled any active bleeding, I clear out the bulk of the blood and clots, and flood the abdomen with abundant hot saline, while in very serious cases the intra-venous injection is at once begun. By proceeding in this manner, I rarely find it necessary to rush matters, although always endeavouring to be expeditious. Having finally cleared out remaining clots, I tie off the offending tube with a continuous ligature. If the outer end of the other tube is sealed, a plastic operation is performed to secure its patency. In one desperate case in the Perth Hospital, in which this procedure was carried out, there was a normal pregnancy eighteen months later. As to drainage, I practically reserve it for septic cases, under which circumstances the abdomen is not flushed out, while iodoform gauze and a rubber tube are brought through Douglas' pouch. Except in such cases the abdomen is left full of saline. When the patient is got to bed the "drip saline" is set going, while the Fowler position is employed in septic cases as soon as practicable.

In some cases I have performed hysteropexy at the same time. Thus, in one hospital patient with pyrexia and left iliac pain dating from a supposed abortion a month earlier, the left tube was enlarged, the uterus retroverted and fixed, and there was a yellow uterine discharge. After curettage, abdominal section disclosed a quantity of old blood clot in the pelvis. The inner portion of the left tube had been gravid, and exhibited two ruptures, one into the peritoneal cavity, and the other into the broad ligament. The outer portion of the tube contained pus continuous with a small abscess in the ovary. After removal of the tube and ovary the uterus was freed and ventro-suspended with excellent recovery. What was the relationship between the gestation and the pyosalpinx? In another case the appendix was removed as well as hysteropexy being performed.

Non-interference.—Operation is not called for in rupture into the broad ligament where the extravasation is aseptic and small. Several cases of this nature under my care have recovered by rest. If the swelling is large or increasing, abdominal section is performed, the clots cleared out, and the cavity closed up entirely, or, after making an opening through into the vagina for drainage, the peritoneum over the cavity is sutured up. Selected cases might be attacked through the vagina, the route of election for septic cases.

The expectant treatment of pelvic hæmatocele gives a low mortality, but the cases have not been followed up on a really large scale, so that it is difficult to be very definite as to end-results. The treatment often requires many months' rest, and may terminate in suppuration with tedious convalescence, or may lead to occlusion of the healthy tube, or to fixation of the uterus in a retroverted position, or perhaps to appendicitis. Obviously, if hæmatoceles were operated on, these evils would be largely avoided. Each case, I suppose, must be judged on its merits; but my personal inclination is to operate in every instance, and preferably by abdominal section, although some advocate the vaginal route. I prefer the abdominal route as more thorough, and therefore likely to be followed by a more satisfactory end-result. With a large hæmatocele increasing in size, or causing pressure symptoms, there can be no alternative to operation;. with a small quiescent hæmatocele, a fair trial may be given to rest. To me, however, the expectant treatment savours too much of trusting to luck; while to operate through the vagina on a large aseptic hæmatocole savours rather of groping in the dark.

(2) The Placental Stage.

In the placental stage the placenta, at any rate after the fourth month of gestation, to a large extent dominates the situation. That operation is essential in this stage all are agreed, but there are different views as to when it shall be carried out with a viable fœtus. Some recommend waiting until after death of the child as the safest course. Others favour prompt operation at all stages of gestation. Pozzi advises operating a little before spurious labour.

Having decided on operation, three methods are to be considered—(1) extirpation, (2) sequestration, and (3) marsupialisation.

Extirpation by abdominal section is the ideal method, and involves no special operative procedures unless gestation has reached at least the fourth or fifth month, when removal of the quick placenta may lead to uncontrollable and rapidly fatal hæmorrhage. With a dead placenta extirpation is relatively safe.

Sequestration of the placenta may be carried out if a quick placenta cannot be safely removed, and there is no sepsis. Even if the placenta is cut through it may still be sequestered if hæmorrhage can be arrested by ligature. In view of the possible supervention of sepsis or hæmorrhage, it is wise to fix the sac to the under surface of the abdominal incision.

Marsupialisation of the sac may be demanded by hæmorrhage or sepsis, although some advise it as a routine measure where the placenta cannot be removed. The practice is commonly followed if the sac cannot be extirpated. In some cases the sac may be sewn up, and the drain carried through the vagina, the route of election for drainage.

When to operate with a viable child.—If operation is postponed until after fœtal death, the difficulty is to decide when the placenta can be safely removed. As a rule the placental circulation is obliterated within two months, but it has been found active a good deal later. Meanwhile there will have been the danger of infection.

Pozzi. as stated, is opposed to waiting until after spurious labour and fœtal death. He thinks the ectopic fœtus has been held too cheaply, many cases being known of its perfect survival; and Lawson Tait speaks of one ectopic child growing into "an elegant woman." All the same, the general belief is that an ectopic baby is unlikely to permanently survive, and that in sacrificing it, we are only forestalling what Nature will almost certainly do if the child is brought alive into the world. If we accept this view, and believe further that obliteration of the placental circulation can be recognised, and that danger during the process is very remote, we will be likely to operate after spurious labour. However, if this waiting is to be our attitude, it will be necessary to be always on the alert in case prompt operation is demanded by urgent symptoms.

Personally, I am opposed to waiting until after spurious labour, mainly on the following grounds:—(1) We have no absolute criteria of complete obliteration of the placental circulation, and if we attempt to remove the placenta earlier, furious hæmorrhage may kill the patient, or demand gauze packing with its attendant risks; (2) at any time after spurious labour infection may supervene; and (3) other accidents may occur, such as rupture of the gestation sac during spurious labour. On the other hand, I favour prompt operation as soon as gestation is recognised. With a viable child there is practically only one great danger—hæmorrhage. If that is escaped, and the placenta can be removed, the patient should commonly make a speedy recovery. To prevent hæmorrhage, Douglas' pouch has been opened, and the mutilated child delivered through the vagina. In similar circumstances the placenta has been cut through after abdominal incision, hæmorrhage being controlled by clamp forceps and chain ligatures. Other measures are gauze packing, and even hysterectomy, the latter being especially demanded in interstitial cases, except where the fœtus has become intra-uterine. Obviously ligature of the main vessels to the placenta before removing it is always the great desideratum.

Although I recommend prompt operation, I realise that before such a rule can be decisively formulated we will require to have answered many questions such as the following:—The proportion of cases operated on after spurious labour in which a diagnosis of obliterated placental circulation is borne out at operation; the proportion of cases in which infection occurs after spurious labour; the proportion of cases in which it is necessary to cut through the placenta to effect delivery; the percentage of cases with a viable child in which the placenta can be removed safely; the mortality in drainage with a live placenta; and the proportion of cases in which a sequestered placenta becomes infected. Until at least all these questions have been satisfactorily answered from a sufficiently large number of cases handled on the lines of the most recent surgical technique, it will be premature to become the standard-bearer of any therapeutic dogma.

Conclusion.

I recommend the following practice, although no surgeon can have had sufficient experience to be an infallible guide:—(1) Operate in ectopic gestation as soon as recognised, except in small broad ligament cases not causing symptoms. Small pelvic hæmatoceles may be treated expectantly, although I favour operation; (2) Employ the abdominal route, except in special cases, when vaginal section may be used, the vagina being also the route of election for drainage.

I might too, suggest the following points for discussion:—(1) Those cases that require prompt operation; (2) Shall a patient collapsed from hæmorrhage be removed from her home for operation? (3) Those cases where expectant treatment may be tried; (4) The indications for the vaginal route; (5) The best method of dealing with the placenta with a viable child; (6) The question of waiting till after spurious labour before operation; (7) Shall a tube in which early gestation has occurred be conserved?

When in 1883 Lawson Tait carried out the first abdominal cœliotomy for ruptured ectopic gestation, he stood forth as the great pioneer in a new field of surgery. Others had believed that the rational treatment of the condition was a surgical operation, but it was reserved for the inspiring genius of Lawson Tait to convincingly put theory to the test of experience. In my last 200 cœliotomies in the Perth Hospital, there were twenty-three cases of ectopic gestation, all except one (an interstitial mole) being operated on at the time of or some weeks subsequent to primary rupture or abortion. Twenty-one cases recovered, although I feel convinced that the mortality in this series must have been exceedingly high but for surgical interference. Since so many lives have been similarly rescued since 1883, there could surely be no honour too great for Lawson Tait's brilliant and beneficent achievement.

DISCUSSION ON ECTOPIC PREGNANCY.

Dr. DUNBAR confined his remarks to two points only—

1. The treatment of cases at the time of primary rupture, and treatment of cases of tubal abortion.
2. Speed in operating.

He advocated the advantages of immediate operation directly the diagnosis was clearly established. In some instances it would be wise to wait until the stage of shock had passed. The condition of the pulse being the main guide, he urged the value of proctolysis by continuous normal saline solution right through the operation directly anæsthesia was established.

The patient was placed in Trendelenberg's position, and Ramsay's apparatus used, as it had the advantage of having a long, soft rubber catheter, was easily kept at 110 deg. F., and at a glance one could see how much of the solution was passing into the rectum. He has used this method recently in all his severe abdominal sections with most satisfactory results in diminishing shock, avoiding collapse, and promoting renal excretion.

Secondly, while extreme care was often necessary when opening the abdomen in these cases to avoid injuring the intestines, which may be displaced by the blood, from the moment the peritoneum was opened by a long incision, the sense of touch *only* was required to find the ruptured or leaking tube of the affected side. Then hold that between the first and second fingers of the left hand, and with the thumb protecting the uterus and any piece of adjacent intestines—which lay in the hollow of a large spoon-like retractor—to clamp the uterine end of the tube. Then rapidly turn out clot, find the ovarian artery, ligature that, and then proceed with less speed to finish the operation.

Dr. R. TATE SUTHERLAND: Every case of menorrhagia should be carefully investigated, and the case diagnosed before rupture takes place.

It is questionable whether patients who are far away from hospital, and who have to be operated upon in unfavourable surroundings, should not be left for a time without operation, and their general condition of health improved by rest; but in cases where there is evidently tubal rupture (not abortion) delay is inadmissible.

If operation is decided upon, the sooner it is done the better, and the first thing to do is to stop the hæmorrhage from the tube before proceeding with operation.

Hæmorrhage is so severe in some cases that patients may die from hæmorrhage in a few hours, as instanced by a quoted case.

Diagnosis has for its object the furnishing of a guide to treatment. We want to know: shall we operate; and if so, when?

The first thing I try to impress on students is: never look for symptoms of pregnancy. Seventeen per cent. of my cases were operated on before a period had been missed, and another 35 per cent. before the end of the fifth week.

Many cases of tubal pregnancy probably go on to tubal mole.

There are two terminations—rupture, and extrusion of the ovum by the ostum abdominal—that require consideration by the general practitioner.

In the one case (rupture) death is imminent, for hæmorrhage proceeds continuously until the patient succumbs; rarely, if ever, do clots plug the vessels.

In the other case the clots plug the tube, and often permanently arrest the flow.

It recurs with their expulsion. The ovum in toto may be expelled, and then all hæmorrhage ceases. I have found three of these cases. In each of these classes there is the rapid pulse, the collapse, and the acute pain.

In the abortion it is, however, shock from distension of the circular fibres. The blue lips, as distinguished from the pallor of hæmorrhage, the mental apathy, and recurrent acute pains generally indicate the difference. In tubal abortion the general practitioner can afford to wait until better surroundings or more expert assistance can be obtained.

With rupture no delay is to be thought about, and operation in any case and by the least expert should be at once undertaken. With regard to the expectant treatment, American results give recovery after fifty-six days, but the adhesions and displacements that form in that time will produce a permanent morbidity.

Surgery should cease to look on saving of life as an ultimate goal.

Dr. T. G. Wilson remarked that no mention had been made in the papers read on the expectant treatment of those cases of ectopic which were first seen when in a condition of profound shock. He had been interested to find that some American authorities advised and practised waiting in this class of case, keeping the patient absolutely quiet and giving small doses of morphia, and not operating till the patient had recovered from the initial shock due to the hæmorrhage. If this method were safe, it might be of service in giving time to prepare for operation in a private house, rather than shifting the patient to hospital, as the jolting in transporting such a patient often tended to increase the bleeding. Personally he preferred to operate at once, as it was impossible to tell how much bleeding was going on, and he thought the sooner the source of bleeding were controlled the better, and once the infundibulo pelvic fold was secured the patient could be stimulated, and intravenous saline used. In the more chronic cases—pelvic hæmatocele due to ectopic pregnancy, rupture or abortion—he was inclined to advise operation rather than wait long for the effused mass to be absorbed, because of the risk of infection and the formation of adhesions. In regard to the diagnosis of these cases of ectopic gestation, he did not hesitate about opening the cul-de-sac in order to be sure about the condition, although one recent writer in the *Australian Medical Gazette* on this subject thought the opening in the cul-de-sac increased the risk of infection. He thought the elaborate washing out of the abdomen that used to be practised in these cases was not at all necessary, and personally he simply cleared out all visible clots and dried the pelvic cavity, and let the peritoneum deal with any blood left.

One point that had impressed him very much in regard to these cases—which are nearly always emergency ones, and have the minimum of preparation for the skin of the abdomen—was the remarkable way in which the wound almost invariably heals by first intention. It raises the question whether the sometimes elaborate preparation of the skin of the abdomen before operation is always a wise procedure.

Dr. Thomas Murphy related a case of normal pregnancy where a large pelvic hæmatocele was apparently due to hæmorrhage from the embryonic placenta, this being situated over the uterine end of the tube. Such a condition might easily be mistaken for dual pregnancy, extra and intra uterine at the same time. He was also of opinion that some of the hæmorrhagic vaginal discharge in cases of tubal gestation was due to back-flow through the uterine end of the tube. In ectopic cases, with symptoms of severe hæmorrhage, as bleeding vessels were the objective he advocated operation without delay, restoratives being applied as bleeding was controlled.

Dr. Chenhall: Tubal pregnancy is a dangerous lesion, since tubal abortion or rupture is inevitable, and the effect appalling. Death may occur from hæmorrhage and shock before aid can be secured Practically all cases afford some warning, which the alert practitioner should quickly interpret. Surgical aid is imperatively demanded in cases of intact tubal gestation. Once diagnosed, abdominal section should be performed as soon as possible after discovery and available skill, condition of the patient, and environment make it safe. Without reservation, I hold this to be the only treatment before abortion or rupture has occurred.

When abortion or rupture occurs, the condition of the patient should alone guide in favour of immediate or deferred operation. Attractive argument may be advanced in favour of immediate laporotomy and stoppage of bleeding, which is threatening death. Such triumphal procedure, aided by physiological and pharmocological support of salines and drugs, will naturally appeal to onlookers, and, if successful, will receive endorsement without that weighty consideration which the question warrants. But it will be admitted by those who have operated on a large number of cases that in probably not more than 5 per cent. has immediate operation been necessary to tie a bleeding vessel. In almost all cases bleeding has practically stopped. The sound surgical axiom, "Secure the bleeding vessel," must, therefore, be modified in its application to these cases. In the first place, a bleeding vessel is rarely present at operation until the clot is disturbed by manipulation, and the superadded shock sometimes blots out the last spark of life. The mortality attending immediate operation is probably not less than 10 per cent. Now, I believe that if 95 per cent. of all cases were operated upon when the condition of the patient is favourable—in which case it may be either an immediate or deferred operation—mortality should be practically eliminated.

Our chief attention should be centred upon this remaining 5 per cent. The chief causes of death following immediate operation are shock, hæmorrhage, and infection from a ruptured tube. Ectopic gestation almost invariably occurs in a tube that has at some time been infected. Before abortion or rupture, this infected area is encapsulated, and hence may be removed in toto. But should reinfection occur at the time of rupture, patients' anæmic condition, with superadded shock, offers little resistance to the invasion of organisms. Moreover, blood left in the abdomen during hurried operation will form a fertile culture medium. Experience teaches us that any plan is best which limits infection to the pelvic cavity, and this is exactly what Nature does. It is thus wiser to defer operation in such cases until patient has attained normal power of resistance, tolerance to local infection, and until the organisms have become less active or inert.

The cases, fortunately few in number, which perplex one most are those showing symptoms and appearance of impending death.

It will be advanced as argument for immediate operation that someone has seen a patient die before anything could be done. Some cases will surely die from sudden rupture, shock, and hæmorrhage, until women become sufficiently enlightened to seek advice earlier, and until practitioners more carefully investigate and interpret the warning notes which almost constantly are sounded as alarms. Again, one occasionally sees death supervene during or immediately after operation, and, although patient clearly dies from superadded shock and not from further bleeding, which has been immediately stopped, it is claimed thereafter that in any case patient would surely have died. These are precisely the cases—the cases on the borderland of death—which, in my judgment, are offered the best chances by expectant treatment; these the cases in which immediate surgical interference blots out the last hope of life. Here calm, well-balanced, conscientious judgment will triumph over surgical technique, however brilliant. It may not win the applause of the multitude, who endorse strokes of genius even if occasionally abortive, but it may save the life of your patient. If patient has just life enough to warrant her being taken to the operating room, let operation be deferred, and expectant constitutional methods be adopted.

In conclusion, I would strongly deprecate the removal of the other Fallopian tube because one has become the seat of ectopic pregnancy.

Dr. BARRINGTON preferred to recognise clinically, and from the operative standpoint, two distinct types of cases:—1. The acute tubal rupture with hæmoperitoneum, marked signs of collapse from blood-loss, and no local physical signs.—The collapse is in proportion to the blood-loss, and rational treatment must look with certainty towards stopping the further hæmorrhage. The more desperate the case while the patient yet lived, the greater the need for immediate action. His practice was to stop the bleeding as quickly as possible by forceps pressure and ligature of the vessels of the ablated portions, and hæmostatic top-sewing of the peritoneal edges. When the bleeding vessels were secured there was no need to "rush matters," for the operation was practically finished when the abdomen was filled with normal saline and the parietes could be carefully layered. The question had been raised: Shall the collapsed patient be removed before operation? He thought much depended on the distance the patient lived from a hospital. If within reasonable distance it should be done, for the improved operative environment had so much to commend it. He had been obliged to deal with cases owing to great urgency in private houses. 2. Less urgent cases with tubal abortion and associated more or less encapsuled hæmotocele.—The signs of collapse were slight or absent, the local pelvic signs marked, and the need for operative treatment was not so imperative, although the sooner carried out the better. He had not found the diagnosis of tubal rupture without difficulty, for it was closely simulated by perforative appendicitis, purulent salpingitis, with patent abdominal ostia and pedicle torsion of a small ovarian tumour. He must admit he had "fallen in" in the diagnosis of such cases. So, too, he had met with difficulty in dealing with mesometric gestations with live placenta. In one case, at the sixth month, tying the feeding vessels failed, the whole placental site continued oozing, and he was obliged to apply gauze pressure, the end being brought out through the cul-de-sac. While the patient recovered, he would in future adopt the suggestion of ligating the cord, sewing up the sac closely, and belating the whole. Reference was made to a specimen present from the Congress Museum, from a case of mesometric gestation beyond term, which, after a stormy convalescence, made a perfect recovery.

Drs. OSBORN COWEN (Kew), FOREMAN (Sydney), HORNE (Melbourne), HOPE LEWIS (Auckland), and TAYLOR YOUNG (Sydney) also joined in the discussion. The PRESIDENT gave a résumé of the papers, and the readers of the papers replied.

ANTE-PARTUM HÆMORRHAGE.

By J. A. CAMERON, M.B., B.S. (Camb.), Ipswich, Queensland.

IN opening the discussion on ante-partum hæmorrhage, time does not permit me to touch upon all the questions that appertain to the subject. I intend to state briefly my own experience, and to endeavour, by comments, and by comparison with and quotations from recognised writers of text-books and articles, to indicate some points for debate or elaboration.

I desire to present the subject from the point of view of the general practitioner, for a severe case of ante-partum hæmorrhage may confront any man in general practice at any moment; time and opportunity may be lacking for him to obtain skilled help, and on his skill, decision, and promptness two lives depend.

From this point of view I have gone through my obstetric notebooks, and among 1,500 consecutive confinements there have been noted sixty-seven cases of bleeding in the earlier months, twenty cases of bleeding during the last three months before, but not during, labour, and seventeen cases of severe dangerous bleeding during labour.

There seems no better classification needed than the orthodox one of—first, accidental hæmorrhage, external, internal, and mixed; and secondly, unavoidable hæmorrhage, with lateral, marginal, and central insertion of placenta. In the first the bleeding is caused by the separation of a normally-situated placenta, and the second of an abnormally-situated one, the blood in both cases coming from the uterine sinuses.

Defining ante-partum hæmorrhage arbitrarily as hæmorrhage from the placental site, during the last three months of pregnancy, or the first and second stages of labour (Barrington, *A. M. Gazette*, June, 1908), there are thus seventeen severe and twenty mild cases in 1,500 labours. While, of course, this includes all dangerous cases and those likely to cause anxiety, it is certain that twenty does not represent the true proportion of milder cases, for I am now persuaded that cases of slight accidental hæmorrhage and partial placenta prævia occur very much more frequently than is usually supposed, and also that severer cases are commoner than text-book statistics would lead one to believe. Owing to the exigencies of a busy practice, it is not always possible to enter up one's notes promptly, so that slight hæmorrhages were possibly often unnoticed or unnoted; indeed, probably they would not always be attributed to the proper cause, for I did not recognise till lately that, as Jardine says in his "Clinical Obstetrics: "When slight hæmorrhage occurs during labour, as it not infrequently does, it probably arises from the separation of a small portion of placenta which has been attached below Bandl's ring."

Although excluded by the above definition, I wish to draw attention for a moment or two to the cases of bleeding during the first six months. Of these sixty-seven are noted; and, of course, no cases that actually aborted are included. Here again I am sure the numbers are too low. As special inquiry has not always been made, many with slight bleeding have undoubtedly been missed. Still, it is interesting to note that in addition to ordinary ante-partum hæmorrhage, practically at least 5 per cent. of all cases have some bleeding during the first six months of pregnancy, some in considerable quantity. In all these sixty-seven cases watch has been kept to see whether the early bleeding of threatened abortion was evidenced at the time of labour by adherent placenta; alteration in size, shape, or situation of the placenta; fatty, calcareous, or other changes in the placenta; hæmorrhage beyond the average amount; labour before the full time; or by any effect on the baby.

My notes state that forty-six had normal labour at term; six are not yet confined; in six premature labour came on; in two it was induced on account of continued bleedings; one had Cæsarean section done elsewhere for some other cause; one had hæmorrhage above the average; and in five others there was some placental abnormality, viz., placenta succenturiata in one case, and adherent placenta on four occasions. This last condition occurred three times in the one patient; on each occasion the placenta was universally adherent, and had to be removed piecemeal under an anæsthetic. There was only one dead baby in addition to the premature ones.

I would suggest that an interesting subject for discussion is opened up by these figures. They are small, but may lead others of riper experience to give their opinions. They tend to show that a considerable number of cases of threatened abortion may be tided over safely and proceed to normal labour at term, and may induce one to refrain from considering an abortion inevitable, and to persevere with expectant treatment longer than, perhaps, is sometimes done.

Again, do some cases of placenta prævia abort in the early months? Possibly both abortions and placenta prævia are due to the same diseased condition of endometrium, but we know nothing of the morbid anatomy or symptoms of diseased endometrium which may cause placenta prævia. Edgar says that most cases of menstruation during pregnancy are due to low implantation of placenta; while Herman, on the other hand, states that most subjects of placenta prævia go through the first half of pregnancy without abnormal symptoms, a statement not supported by my few figures, for five, or nearly half, of the cases of ascertained placenta prævia had some bleeding during the second and third months.

Reverting to the possibility of slighter cases of both accidental and unavoidable hæmorrhage being overlooked, twenty others are noted as bleeding more or less severely during the last three months, but not much at the time of labour. These are presumably slight cases of one or other condition, probably generally placenta prævia; but they remain without definite diagnosis, as the bleeding ceased before labour began, and did not recur to any extent then. In such cases examination of the placenta and membranes after labour may assist in forming an opinion.

Sir Arthur Macan, in his address at the British Medical Association meeting in 1904, says:—"There can be no doubt that a small amount of hæmorrhage between placenta and uterine wall is by no means uncommon. . . . In many cases the diagnosis is impossible till after expulsion of the placenta. Hence a great many cases of slight accidental hæmorrhage escape our observation." Munro Kerr, in "Operative Midwifery," says:—"Not a few of these cases (that is, lateral forms of placenta prævia) are never recognised; bleeding occurs, but with the separation of the small portion of the placenta attached to the lower segment it ceases, and the child is born. If the membranes are examined after birth it will be found that the rent is close to the margin of the placenta."

There remain seventeen cases of severe or dangerous ante-partum hæmorrhage, of which ten were certainly, and three very probably, cases of placenta prævia; and two certainly, and two probably, cases of ante-partum hæmorrhage. Of these, three of the mothers died as a result of the hæmorrhage and shock.

Diagnosis.

Five of them being classed as probable, at once opens up the question of diagnosis. Theoretically, from the point of view of carrying out efficient treatment, accurate diagnosis is essential; actually and practically, differentiation between the two conditions is not always essential, or even possible. I summarise a few opinions in support of this view. Jardine, in "Clinical Obstetrics," says that sudden, apparently causeless, hæmorrhage, slight or severe, from six months onwards must be placenta prævia or accidental hæmorrhage, except in a few obvious cases, such as injury to the genitals, ruptured varicose veins, &c. The history of the onset does not help; one has to depend on examination. If the cervix admits a finger, as it generally does, and you can feel the placenta, it is placenta prævia. If you cannot reach the placenta by sweeping the finger round the lower uterine segment it is probably accidental hæmorrhage. If you cannot get your finger in you cannot be absolutely sure. Some profess to be able to tell by abdominal palpitation. Jardine says he cannot, and never will be able to do so, an opinion most of us would endorse for ourselves. Sir Arthur Macan, in the address already quoted, says:—"If we feel the placenta the diagnosis is made, but we are not justified in concluding that, if the placenta is not felt at the os, the hæmorrhage is not due to low insertion of the placenta. I am of opinion that it is not by any means always possible to make this diagnosis. I confess that I do not know how to diagnose hæmorrhage due to low insertion of the placenta from external accidental hæmorrhage, but it is not likely to be confounded with internal accidental hæmorrhage, or even with combined hæmorrhage." Dr. Barrington, in his excellent article in the *A. M. Gazette*, June, 1908, says:—"A definite diagnosis as to which exists can only be made with certainty by digital examination through the internal os." Munro Kerr, in "Operative Midwifery," says:—"In coming to a diagnosis one must depend on the vaginal examination, and actually feel the placenta with the examining finger. If one feels the placenta one calls the condition placenta prævia; if one does not feel it, one diagnoses it accidental hæmorrhage." Again, in discussing accidental hæmorrhage, he implies that cases of placenta prævia, with only a small portion of placenta attached to the lower uterine segment, are overlooked and mistaken for external accidental hæmorrhage. "Fortunately," he adds, "treatment differs but little in the two conditions." What, however, is of the greatest importance, as he forcibly points out, is to recognise mixed forms of accidental hæmorrhage, and not to make the mistake of measuring the seriousness of the case by the amount of evident bleeding, but by the general condition of the patient and the state of the uterus. So we arrive at the conclusion that most cases can be readily diagnosed by digital examination, but that differentiation between slight lateral placenta prævia and external accidental hæmorrhage is not always easy, or of great moment from the point of view of treatment.

Frequency of Occurrence.

The next point I would bring forward is the frequency of occurrence. Here, in a series of 1,500 consecutive cases, are thirteen severe cases of placenta prævia, and probably several milder ones as well. I have been able to obtain very few figures from records of private practice for comparison. Dr. Cowen, of Eaglehawk, Victoria, records 700 consecutive cases, with three placenta prævia; Dr. Butler Walsh, of Victoria, four in 907 labours; Dr. A. H. Hamilton, of Adelaide, seven in 1,000 labours; and there were also seven in 1,000 in some figures published in the journal of the American Medical Association in 1908. Hospital figures should show a higher percentage of occurrence, yet they invariably make it lower. Glasgow Maternity Hospital gives it as 1 in 300 cases; Berlin Klinik, 1 in 220; and the Polyklinik, 1 in 300; the Boston Lying-in Hospital, in 6,700 labours, 1 in 239; Edgar's text-book, 1 in 244 in 2,200 pregnancies; while many authorities show a still lower percentage—for example, Norris and Dickinson, in "American Text-book of Obstetrics," 1 in 1,000 to 1 in 1,600; Herman, in "Difficult Labour," gives 1 in 1,000; Galabin, 1 in 534 in 49,845 deliveries; and various German authors put it at 1 in 1,560.

Accidental hæmorrhage is stated by nearly all authors to be rather more frequent than placenta prævia—e.g., eighty-three severe cases in 17,200 deliveries at the Rotunda. My small series make it to be considerably less frequent—four as against thirteen.

Mortality.

The mortality in placenta prævia is variously given from about 4 to 25 per cent. for mothers.

The best figures I know of are those of the Rotunda, published last year. During the previous twenty years there were 32,546 confinements, with 138 cases of placenta prævia, of which five died, or a mortality of 3·6 per cent.; and all of these, except one, were moribund or in a state of advanced sepsis on admission. The treatment adopted is bipolar version, and leaving expulsion to Nature. It is universally agreed that the maternal mortality of all cases treated by bipolar version is from 4 to 8 per cent.; and it is difficult to see how this can be much improved upon. The fœtal mortality is from 50 to 60 per cent. in cases so treated, and a considerable proportion of these born alive are premature, with lessened chances of survival. In accidental hæmorrhage, the maternal mortality ranges from 4·9 per cent. in eighty-three Rotunda cases (of which forty-three were treated with plugging, with two deaths), and 11·4 per cent. from the hæmorrhage alone at Guy's Charity, to nearly 51 per cent. in 106 cases of concealed hæmorrhage collected by Goodell. On the latter figures Jardine comments that the mortality is too high for all cases and too low for concealed hæmorrhage only. The fœtal mortality is from 60 to 94 per cent. In Goodell's list only six of the children lived.

The death of the child is due not so much to the loss of blood as to failure in elimination of carbon dioxide.

Apart from actual mortality, the morbidity rate is higher than in normal labours.

Treatment.

Prophylaxis.

Little can be said on this subject. A few facts stand out. First, the much greater prevalence of both conditions in multiparæ. In my figures, all the seventeen severe cases were multiparæ, and sixteen out of the twenty milder cases. Secondly, that previous labours have often been abnormal; this was so in eleven at least out of the seventeen—two figure twice in the series. Thirdly, the previous general health of the majority was indifferent; four were very stout and flabby; two had albuminuria; three others were previously weak. sallow, and anæmic; and three others had pelvic trouble previously. In an interesting and suggestive paper in the Journal for 17th June of this year, Davies-Colley publishes a series of nine cases which he thinks furnish conclusive evidence of the causal relationship between the toxæmia of pregnancy and accidental hæmorrhage. The probability is that in all cases there is some disease of the decidua also; but beyond this I do not think one can say anything definite at present. There is therefore little to base any prophylactic treatment upon. Edgar sums it up tersely: "There is no preventive treatment of this condition."

Actual Treatment.

Before discussing the actual treatment, I will give brief notes of the three fatal cases in my series, as they illustrate the dangers to be combated. The first was a stout multipara, seven and a half months pregnant, and the first case of severe ante-partum hæmorrhage I had ever encountered. Excessive bleeding came on immediately after sustaining a heavy jolt while driving home over a rough road. Labour proceeded forthwith, the placenta was felt easily with the finger, and seemed partly detached; she was safely delivered, and seemed well and the danger past, when, about three-quarters of an hour after delivery, she began to be restless, tossed about, got weaker and weaker, and died one and a half hours after labour was completed; the uterus was well retracted, and no further bleeding had occurred.

The second, also a stout multipara, suddenly had a free bleeding about two weeks before term. This was in the evening, and as she lived a long way out of town she did not send in till next morning, as no more bleeding and no pains came on during the night. Next morning I saw her. The cervix was not dilated at all, nor taken up, and was difficult to reach; no part of the fœtus could be felt, and the finger could not enter the external os. She refused to be moved into town, and, although she promised to stay in bed, after I left she got up and did her day's work. In the evening she suddenly became faint, and collapsed, and died in twenty minutes, undelivered, of course. Only a very little blood came from the vagina. There was no post-mortem. It is hard to see what would have been the best treatment for this case.

The third case was also a multipara, in poor health. At the end of the eighth month, after a heavy day's washing, she had a sudden most profuse hæmorrhage, leaving her blanched, almost pulseless, and with feeble respiration. Labour pains came on, the membranes were ruptured, and she was delivered with forceps after a while. She only lived a few hours, and never rallied under restorative treatment.

What is the best line of action? A hard-and-fast rule for all is absurd; yet, from the point of view of the general practitioner, who generally has to attend his confinements by himself, and perhaps in unfavourable surroundings, a definite simple plan of action is desirable, such as can be carried out single-handed and with the simplest apparatus possible.

Dr. W. S. A. Griffiths, of St. Bartholomew's Hospital, at the annual meeting of the British Medical Association in 1906, in the discussion of the treatment of accidental hæmorrhage, said:—"The treatment of accidental hæmorrhage should not be considered apart from placenta prævia." And I want to take up that view as much as possible.

For the present, postpone the consideration of grave cases of concealed accidental hæmorrhage, very rare and very fatal. For the rest, the first question that arises is: Is there any justification for temporising, once a hæmorrhage has occurred? One may arrive at a patient and find that there has been considerable bleeding, which has stopped. The temptation is strong to advise rest in bed, and to tell the nurse to send if bleeding recurs. This is wrong.

My second fatal case recorded above emphasises this point. When a severe hæmorrhage has occurred the uterus should be emptied without fail and without delay; at all events, if placenta prævia has been diagnosed. As Herman puts it: "As soon as ever placenta prævia is found out, be the bleeding great or little, labour ought to be brought on. The patient is not safe from hæmorrhage until she is delivered." And: "There is no treatment, except delivery, which will stop hæmorrhage in placenta prævia." This opinion seems inconsistent with the fact that I have recorded twenty cases of hæmorrhage during the last three months of pregnancy, in which nothing was done, and no harm followed, and that I have considered mild cases of both conditions to be commoner than generally believed; still, as a general rule, it is the safe one to follow. The chances of a living child are thereby diminished, but even by waiting they are not rosy.

Treatment may be summarised thus:—In placenta prævia, if the cervix is dilated, do bipolar version, bring down a leg and wait, watchfully, for Nature to complete delivery; if the cervix be undilated, proceed to dilate with fingers, bag, or plug, and when dilated carry on as before. In accidental hæmorrhage, if the os is dilating and the pains are vigorous, rupture the membranes, and possibly give a dose of ergot, and apply a tight abdominal binder. If the os is not dilating, choice must be made between dilating with a bag, instrument, or fingers, and extracting by the quickest method, thorough plugging of the vagina, and Cæsarean section.

In either condition the presence of pains and some dilatation of the os make the task simpler; bipolar version is done, unless possibly the dilatation is so far advanced that forceps may be applied, and a leg is brought down. By this means the bleeding is largely controlled; labour is then allowed to proceed naturally, no attempt being made to hurry or drag the child through, this being vitally important in the interests of the mother.

If, however, there is no dilatation, and there are no labour pains, the membranes being unruptured, what then? In the vast majority of cases of both conditions plugging of the vagina efficiently will prove sufficient. The patient is put in the lithotomy position, a little chlorofrom having been given; then, with strict precautions against infection, the vagina is plugged in the manner described by Colcough in an excellent article in the "Journal of Obstetrics and Gynæcology of the British Empire" for June, 1902. In placenta prævia a piece of iodoform gauze or bismuth gauze is inserted into the cervix if possible, then in both pads of absorbent wool an inch or so in diameter, wrung out in some antiseptic fluid, are packed into each fornix and round the cervix, and then in layers till the whole vagina is full; a tight binder is fastened on and a large perineal pad fastened tightly fore and aft to the binder, thus exerting firm pressure on the vaginal plug. This is the safest course in placenta prævia when the patient is not in labour and the cervix will not admit two fingers, and is recommended by almost all British obstetricians. The plug should be removed and the vagina repacked after some six hours, if necessary, till labour has progressed enough to allow of bipolar version.

What alternative is there to this procedure? Simple rupture of membranes, or partial forcible dilatation of the os, and the introduction of de Ribes' or other bag. Undoubtedly the bag can be introduced in a small margin of cases before bipolar version could be done, and by many it is favoured as the best line of treatment. Authorities can, as usual, be quoted for and against. Dr. R. Warren, of the London Hospital, in the *Lancet,* 3rd November, 1906, gives forty cases of placenta prævia, ten of them severe, treated with de Ribes' bag. None of the mothers died. He says: "The bag, once introduced, never failed to control the flooding and to induce labour, though the latter was at times a lengthy matter. It seems equally good whether pushed through the membranes or between the ovum and the uterus." But he goes on to say: "After the bag is delivered, unless the head or breech is driven down, internal version is the best course, since flooding occurred in several cases after the bag was expelled. In these cases the insertion of the arm for version acts as a plug till the fœtus is turned, and can in its turn act as a plug." This double admission, to my mind, gives the case for the bag away. First, it may not be any speedier in bringing on labour; and secondly, another handling of the patient is sometimes necessary to stop the bleeding, and, from the point of view of simplicity, it necessitates keeping, and keeping in order, another instrument which can equally well be done without, and which obviously is very seldom required. As an example of an opinion opposed to Warren's, I quote Fothergill, who says: "It is very doubtful, however, whether rupture of membranes, dilatation by de Ribes' bag, and extraction by forceps is as safe a course for the mother as packing the vagina and bipolar version." Also by Dr. Barrington, in his article already referred to, says: "For use in general practice it (de Ribes' bag) is inferior to version; but in the rare cases where the membranes have been ruptured, and turning is no longer possible, it may be of great service as a preliminary to slow forceps extraction."

Turning now to accidental hæmorrhage cases, where labour is not advanced far enough to allow of hastening delivery, or where the patient is suffering from loss of blood and shock, opinions are not so unanimously in favour of plugging as above described. On the one hand, all the Irish obstetricians support it unreservedly. It is pointed out that further bleeding is effectually checked, as a rule, and that if the patient is much shocked or collapsed from loss of blood time is gained for recovery to some extent before the additional strain of labour, with the possibility of post-partum loss, and during this internal saline transfusions and morphia may be given. Sir Arthur Macan, in his paper on this subject at the British Medical Association meeting already mentioned, pointed out that four consecutive masters of the Rotunda favoured plugging; and he propounds this summary of his views for discussion: "Have we, in systematic plugging the vagina, combined with firm pressure on the uterus and a strong perineal bandage, a safe, simple, and reliable method of treating all cases of accidental hæmorrhage?"

He states that no absolutely satisfactory treatment had been devised for accidental hæmorrhage, but he considered systematic plugging of the vagina was best. He adds: "This method will simplify treatment of accidental hæmorrhage greatly, and save the lonely practitioner much mental and moral anxiety, and preserve many lives." He points out that, as an alternative to this method, one may dilate the cervix manually or instrumentally, or adopt vaginal Cæsarean section, and adds these pregnant words: "But no method of dilatation gives an answer to the question: How long have I got to effect dilatation of the soft parts before the patient dies of hæmorrhage? or, How long will the soft parts require for dilatation without risk of producing serious rupture of the cervix? Unless we adopt the method of treatment by plugging, these questions must remain for ever the unsolved difficulties in the treatment of bad cases of accidental hæmorrhage." I have quoted these views at length because they express exactly the points of view I believe to be best for the average man dealing single-handed with these critical cases.

The alternative of vaginal Cæsarean section I have briefly touched upon later.

It is right now to point out that the British obstetricians, on the other hand, are not so unanimously in support of the plug. Some, notably Jardine and Herman, condemn it severely; but, as far as I can make out from their books, they do so without stating very definitely why, and if the Dublin practice gives good results theoretical objections fail to convince. The weak point in the method is pointed out by Fothergill, who, by the way, favours it on the whole. If the pressure inside the uterus equals the blood pressure, no further hæmorrhage can take place into its cavity, and the pressure inside the uterus is raised by plugging the vagina very tightly, and applying a tight binder; but, to ensure this, the uterus must be healthy, the ovum intact, and the plug tightly applied. The healthy condition of the uterus is possibly the lacking factor. He goes on to say that concealed accidental hæmorrhage can only be extensive if the uterine wall is diseased; while some, notably Edgar, go so far as to say that it is questionable whether accidental hæmorrhage can ever occur with a healthy placenta and uterus. In actual practice, however, one will undoubtedly find that the plug, firmly applied as I have described, does check bleeding and allow time for recovery on the part of the patient. Often, as in Colcough's description, when the plug is removed, which is done when pains are progressing satisfactorily and the plug beginning to bulge, the cervix is found well dilated, and labour terminates speedily, or may be assisted by bipolar or podalic version, or the use of forceps, according to the extent of dilatation of the os.

Be it remembered always that the risks in these cases are chiefly three—(1) Hæmorrhage; (2) shock; and (3) sepsis. I think the treatment I have advocated will be found effective in controlling hæmorrhage, less causative of additional shock, and certainly not more provocative of sepsis. Persons who have lost much blood are much more susceptible to septic infection than others, and this must not be lost sight of amid the natural excitement caused when suddenly called upon to deal with a big hæmorrhage. Three aids in combating the subsequent shock must not be forgotten; they are a little chloroform when manipulation is in progress, a hypodermic of morphia, and saline transfusions beneath the skin or in the rectum after labour. The rectum of a recently-delivered woman absorbs fluid very readily; and raising high the foot of the bed on chairs speedily carries out an autotransfusion.

Be it remembered, also, that very little post-partum hæmorrhage may turn the scale if there has already been a big loss, so that one must be ready to deal with this promptly. The placenta should in both cases be removed by the insertion of the hand if it does not soon follow the baby, and measures adopted to check bleeding as much as possible. An additional advantage of vaginal plugging is that post-partum hæmorrhage probably does not occur if sufficient time elapses between its adoption and the delivery.

It will have been noticed that no mention has been made of the use of metal dilators of the Bossi or Frommer type. In my opinion they are bad, or at all events not necessary; both forcible dilatation and forcible delivery are to be avoided.

There remains to be considered the few and terrible cases of concealed internal hæmorrhage.

I cannot speak from any personal experience of these. The possibility of confusion with rupture of the uterus is always mentioned. The latter condition is usually in evidence late in the second stage of labour, while the continuous rending pain and hard condition of the uterus of concealed hæmorrhage are characteristic, and come on early. The concensus of expert opinion seems to be that these and the worst mixed cases, in which the mortality is well over 50 per cent. at present, are best dealt with by Cæsarean section, abdominal or vaginal, the alternative being dilatation by hand or instrument, and delivery by version or forceps. Munro Kerr, in his book "Operative Midwifery," advocates section, but two cases he so treated both died a few hours after. In Colcough's series, one treated by the method of Porro's operation died, while three treated by plugging recovered. Evidently, from his paper, the feeling he has is that even in these cases a good deal is to be said for the plugging method. If so, it is again satisfactory from my point of view, for obviously most men are not sufficiently skilled to successfully carry out such an operation as Cæsarean section on a patient already in a critical condition, and are often debarred from procuring such expert aid as would be necessary.

Obviously, if operation is to be successful, it must be tried before, and not after, the patient has been further reduced by other methods having been unsuccessfully tried. I feel if, for instance, the second of my fatal cases could have been treated by operation when I saw her, and before her second hæmorrhage, her life might have been saved; but even in this case it would require strong convictions on the part of the doctor to insist on, and strong faith on the part of the patient to agree to, the prompt application of such a heroic procedure.

There seems no need for Cæsarean section in the treatment of placenta prævia.

In conclusion, I beg to submit the following points as, among others, worthy of discussion:—

(1) The after-history of patients who have bleeding in the early months.

(2) The part of placenta prævia in causing early abortions.

(3) The possibility of discovering from the histories of patients whether any diseased conditions could be defined, such as would lead one to suspect the possibility of ante-partum hæmorrhage, and to adopt prophylactic measures.

(4) The frequency of occurrence of both mild and severe cases of both conditions.

(5) The question of treatment; can it be improved from the point of view of reducing the fœtal mortality? Does any routine other than I have suggested offer a better percentage of recoveries for the mothers?

POST-PARTUM HÆMORRHAGE.

By J. W. Dunbar Hooper, M.D. (Durham), M.D. (Melb.).

Formerly Hon. Surgeon to the Women's Hospital, Melbourne.

I desire to thank the Committee of this Section who did me the honour to ask me to bring the subject of post-partum hæmorrhage under your notice to-day.

Unquestionably post-partum hæmorrhage may be one of the most alarming occurrences with which a medical practitioner may be called on to deal. Such an incident, serious enough in a city where professional help is quickly obtainable, may become a tragedy in a bush practice where the medical attendant has to act without skilled assistance. This fact alone impelled me to accept the invitation of our committee, and I earnestly hope that my presentation of the subject to you, and the subsequent discussion, will arouse your keen interest, and enable us more successfully to combat an obstetrical emergency which may be nothing short of appalling.

Post-partum hæmorrhage—obvious or concealed—may be defined as "loss of blood, after delivery, from any part of the parturient canal, sufficient to produce signs and symptoms of faintness, collapse, or death." Crile very clearly distinguishes shock from collapse. In severe post-partum hæmorrhage we have to deal with collapse in its most serious form.

I believe that many members of this Section, when they read the titles of the subjects for discussion, exclaimed, "Ah! *we* never see a case of post-partum hæmorrhage nowadays." Is it really so? We may not, owing to extreme care, or by that immeasurable quantity called luck, have actually lost a life from post-partum hæmorrhage, but surely each of us has had *extreme* anxiety when such a case has presented itself.

Post-partum Hæmorrhage.

Hæmorrhage after delivery may come from various sources. Hæmorrhage from the placental site is, however, so much the most frequent and most important that this is regarded as post-partum hæmorrhage *par excellence.* The subject is one of immense importance, for post-partum hæmorrhage is one of the most dangerous complications of parturition. It may occur after the most perfectly normal labour, and a household may thus be unexpectedly plunged into grief by the sudden death of the patient. Nor is there any emergency in which so much depends upon the care and skill of the physician. The occurrence of hæmorrhage at all may generally be prevented by a careful and correct management of the third stage of labour. When hæmorrhage does occur, the life of the patient will generally depend upon the promptitude and vigour of the treatment.

Office of the Victorian Government Statist,
Records Office, Queen-street, Melbourne, 3rd April, 1911.

Sir,

In compliance with Dr. McLaren's request I have the honour to state that the numbers and ages of women who died from post-partum hæmorrhage in Victoria, and the number of births in each year (1907–8–9–10) were as follow:—

	1907.	1908.	1909.	1910.
Births	31,369	31,101	31,549	31,437
Deaths from post-partum hæmorrhage	14	8	14	19
Ages at death (years)—				
15–20	1	...	...	1
20–25	2	3	...	2
25–30	3	1	4	3
30–35	1	...	...	4
35–40	7	2	7	7
40–45	...	2	3	2

In four years in Victoria, 1907–10, inclusive, 55.

I have, &c.,
A. M. Laughton,
Government Statist.

Dr. Dunbar Hooper,
Collins-street, Melbourne.

Now, to show you that even of recent years the actual mortality is not a negligible quantity, and without at all concerning ourselves about the morbidity, let us consider the figures which have been given to me by the Government Statisticians in the different States and New Zealand:—

(2) *Tasmania.*

From 1890 to 1910, inclusive.

Total number of births	105,015
Total number of deaths from post-partum hæmorrhage and flooding	31
Including abortions	156

(3) *Victoria.*

From 1890 to 1910, inclusive.

Total number of births	681,185
Deaths from post-partum hæmorrhage, including placenta prævia and flooding	517
The total number of deaths of all women in childbirth (but this would include sepsis and accidents) ...	2,627

(4) *New South Wales.*

From 1890 to 1910, inclusive.

Total number of births	823,266
Deaths from post-partum hæmorrhage alone	619

(5) *Western Australia.*

From 1891 to 1906, inclusive.

Total number of births	36,663
Death from parturition, including accidents of childbirth	297

(6) *Queensland.*

From 1906 to 1910, inclusive.

Total number of births	15,554
Deaths from post-partum hæmorrhage	73

(7) *South Australia.*

From 1890 to 1910, inclusive.

Total number of births	192,737
Deaths from post-partum hæmorrhage, including accidents of childbirth	373

(8) *New Zealand.*

From 1890 to 1910, inclusive.

Total number of births	441,490
Total number of deaths from post-partum hæmorrhage, placenta prævia, and other accidents of child birth (not including abortions)	915

Under ordinary circumstances we know that hæmorrhage is controlled by the retraction and curling up of the middle coats of the torn arteries, and by the coagulating power of the blood as it is poured out, coming into contact with surrounding tissues. In the uterus there is a special arrangement of the muscular fibres interlacing around and among the vessels and sinuses, which fibres contract and retract during the third stage and afterwards.

It is said that the human organism can withstand a loss of blood equal to 3 per cent. of the body weight without death ensuing, and it has been stated that, on the average, 6 to 8 ounces of blood are lost at a normal confinement.

What are the effects of severe hæmorrhage?

Firstly—Syncope, followed by temporary cerebral anæmia and recovery.
Secondly—Cerebral anæmia.
Thirdly—Collapse, followed by recovery.
Fourthly—Sudden death—within an hour of delivery.

Among the aborigines of Australia—in reply to my letters sent to heads of the Public Medical Service in each State, full reports have come to hand—death from post-partum hæmorrhage is extremely rare; in fact only two cases are known, and they were unattended in their confinement.

(My hearty thanks are due to Dr. Ashburton Thompson, G.M.O., also to Dr. Ramsay Smith, for their trouble and courtesy in supplying this information.)

Department of Public Health, Wellington, New Zealand.

[Copy.]

Sir,

I have the honour to acknowledge receipt of your letter of the 23rd June, asking if there are authentic instances of Maori women dying in labour from post-partum hæmorrhage, whether attended by medical men, or midwives, or unattended.

In reply I have to state that Dr. Pomare, Medical Officer to the Maoris, informs me that he has looked through his records and finds no cases of death from post-partum hæmorrhage among Maori women, and that he has known personally of only one case.

Other enquiries made, though perhaps indefinite, enable me to state that I think you would be quite safe in saying that post-partum hæmorrhage among Maori women, whether attended by medical men or midwives, or unattended, is extremely rare.

J. P. Frengley, M.D.,
Medical Secretary.

It is surely a striking fact that post-partum hæmorrhage among mammalia is almost unknown, and death from post-partum hæmorrhage of one of the mammalia having a placentation similar to that of a human being is extremely rare; indeed it is practically unknown for one of the domestic or wild animals to die from that cause, unless there has been *trauma*.

Among the mammalia the uteri conforming most closely in structure to those of women are found in the primates and Macacus monkey. These can be seen and studied in the Veterinary School of the Melbourne University. In these animals the arrangement of the placenta and fœtal membranes occurs as in women. In the cow, the duration of gestation being exactly that of a woman, the placenta is in a series of forty or more cotyledons, any one of which may be detained in utero, and perhaps give rise to sepsis. The placentation in a mare is annular or circular, with fœtal membranes only, yet death from post-partum hæmorrhage either in cow or mare (unless due to trauma) is practically unknown. I am assured by Professor Gilruth, Dr. Stapley, and Dr. Kendall, and many breeders of stock, who have had enormous practical experience as veterinarians amongst cattle, that they have never even heard of any mares, sheep, or cows dying from post-partum hæmorrhage, unless there had been some distinct trauma.

Further, I have inquired from the Directors of the different Zoological Gardens throughout Australia, and not one can tell me of severe hæmorrhage following parturition—not even in the monkeys—nor has a death from hæmorrhage ever been reported. Possibly the position taken up during the act of parturition has an important influence in checking hæmorrhage in the mammalia.

I believe that the mental anxiety induced in most civilised women by parturition, and the unskilled or prolonged use of anæsthetics are potent factors in the causation of uterine atony and consequent hæmorrhage.

Is there anything in the anatomy of the parturient which should indicate the possibility of severe hæmorrhage?

Turning aside for a minute to the uterus of the rabbit, we find that, owing to the strong contraction of the muscles of the uterus at the time of the birth of the young, the bare patch from which the placenta has been separated is at once gradually reduced in size, while the loosely attached mucous membrane of the placental region slips over it and closes the wound almost immediately. We find nothing analogous to that in human anatomy or physiology. The woman has to rely on the proper functioning of the interlacing muscular fibres at the attachment of the placenta to the uterus, and her uterus separates and detaches the placenta from itself, and not *vice versâ*.

In woman, immediately after childbirth, part of the inner surface of the uterus has a large surface wound, since by the detachment of the placenta and the deciduæ, numerous blood vessels are ruptured. Also, during the first days of the puerperium, fragments of the spongy layer of the deciduæ veræ and serotina, which remain at birth, continue to be detached from it. Only the deepest layer of mucosa, that which immediately comes into contact with the musculature of the uterus, is retained. This still contains remnants of the cylindrical epithelium of the uterine glands.

This, according to Hertswig, and more recently in Kybel and Mall's Embryology, is portion of the anatomy of the placentation, and on it we should base our treatment of the third stage of labour. It is possible that by neglect in applying the knowledge obtained from the study of the anatomy of the third stage we render our patient more prone to post-partum hæmorrhage. Obviously, Nature must and does apply some method by which she closes, obliterates, or covers the raw surfaces so as to prevent such a hæmorrhage from it as would grievously injure the woman.

Jellett states that during the third stage, and subsequently, there is contraction of the muscle fibres around the vessels, then retraction of those muscular fibres.

Research has been made at my request through the journals and reports of the Royal Society of Medicine of London, and nothing more recent has been published with reference to the Pathology of post-partum hæmorrhage, and the pathologists of some of the largest hospitals say the same—just as Jellett puts it.

Gooch, Matthew Duncan, and R. A. Gibbons, all report cases where *incomplete* tonic spasm of the uterus, without retention of placenta or membranes, was followed by severe post-partum hæmorrhage.

On reading the most recent literature on placentation the most significant fact is that any retrograde change or active cell-growth in the fœtal epiblast of the chorionic villi, as might occur during or after gestation, would lead to such an alteration of tissue in the neighbourhood of blood vessels as would prevent the closure of the latter when torn. Such a condition is known to occur, as in chorio-epithelioma, and to cause such hæmorrhage as to necessitate complete hysterectomy. This chorio-epithelioma invades the muscle below the mucosa more or less deeply, and so injures the musculature as to damage its contractile power.

It has been stated and proved by Webster, and Teacher, that the cells tend to grow actually within the lumen of the uterine vessels, and so cause hæmorrhage in the early stages of the disease.

There are, therefore, I take it, five factors in the causation of post-partum hæmorrhage:—

Firstly—Some abnormal condition, local or general, in the patient herself, organic or functional.

Secondly—Some error in the working of the normal mechanism which controls hæmorrhage after parturition.

Thirdly—Dystocia, causing a prolonged second stage and uterine exhaustion.

Fourthly—Traumatism.

Fifthly—Post-partum hæmorrhage may be due to paralysis of the site of the placental insertion, with firm contraction of the remainder of the uterus. (Muller's "Handbusch," edited by Veit. Vol. II, page 121.)

In the *Lancet* for 1906 (page 127), Schroeder and Winckel are quoted as reporting a case of post-partum hæmorrhage due to lacerations between the clitoris and the orifice of the uretha. The patient died.

Professor Whitridge Williams (Text Book on Obstetrics, second edition, 1910) considers "the most common cause of post-partum hæmorrhage to be retention of the partially separated placenta, or of individual cotyledons, less often deep tears involving the birth canal, or, in very rare instances, to defective functioning of a uterine musculature—that is, atony."

In an experience which covers twenty-five years of continuous practice, I should put the causes of post-partum hæmorrhage in exactly the reverse order, and I am compelled to regard the teaching in some of the well-known text-books on obstetrics on this point as misleading. And, if our view of the causation of this serious condition has been wrong, it follows that our treatment has been wrong. Professor Williams admits that as long as pieces of placenta remain firmly attached to the uterine wall the possibility of post-partum hæmorrhage is slight, but he says that when a piece has become "*partially* separated, normal action of the musculature is interfered with, resulting in the torn vessels of the partially denuded area remaining uncontracted, and hæmorrhage follows." I hold that if there were no atony of the uterus, if the uterine musculature were neither diseased nor fagged nor unable to contract, this area—whether denuded partially or completely—would be compressed. He blames the too early and too energetic use of Credé's method, and he says distinctly—

> "The retention of isolated cotyledons, or of a small succenturiate lobe, interferes with the normal contraction and retraction of the uterus."

Here I, with the greatest respect, beg to differ from him. On those words, found in many text-books, the whole treatment of post-partum hæmorrhage has been based for many years, and each succeeding generation of students has accepted them.

Veit absolutely denies the possibility of primary atony, thereby ignoring the fact that clinical experience has proved, and is supported by pathology, that atony of the uterus has caused death within a few minutes from post-partum hæmorrhage without any retention of placenta—wholly or in part—as proved by photograph of specimens borrowed from the Pathological Department of our University, through the courtesy of Professor Allen.

What are the general conditions observable in a patient which would lead one to anticipate the onset of post-partum hæmorrhage?

First of all the age of the patient, associated with arterio-fibrosis at 40 and upwards, frequent child-bearing, dystocia, the alcoholic habit, syphilis, goitres, myomata, chronic nephritis, yellow atrophy of the liver, myxœdema, and hæmophylia, hydramnios, and gestation of twins. Also prolonged anæsthesia in the second stage, especially when chloroform is administered in a small ill-ventilated room, near a burning gas jet.

In the *Practitioner*, Vol. I, 1899, this subject is fully discussed and its dangers described. I believe that scopoliamine-morphine narcosis tends to increase post-partum hæmorrhage.

N.B.—A hurried or mismanaged third stage is the most potent factor in the causation of post-partum hæmorrhage.

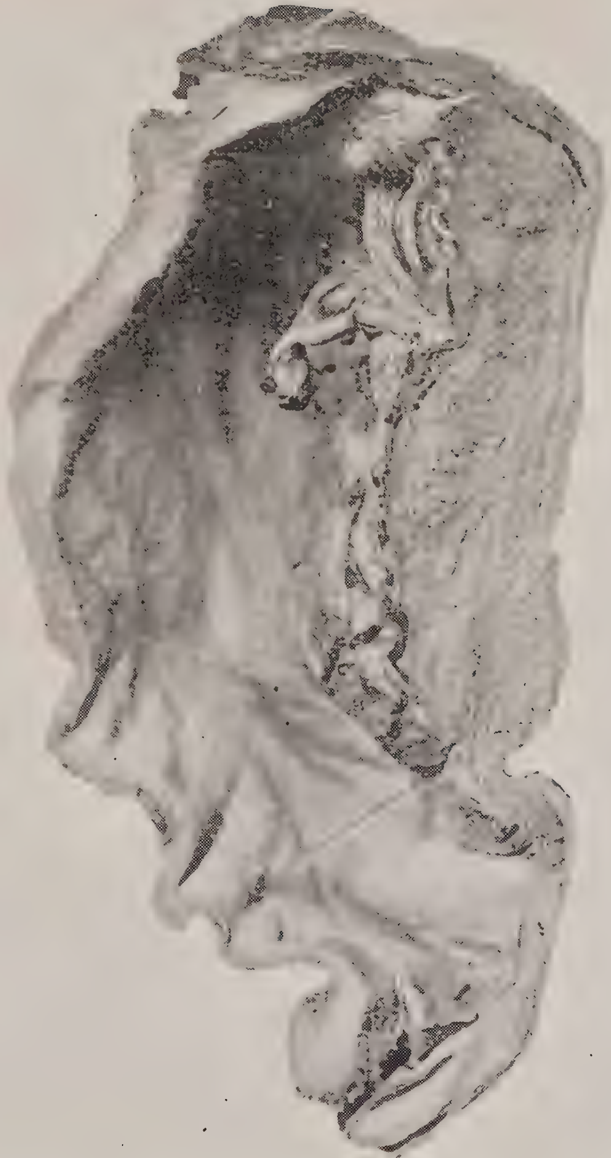
The source of post-partum hæmorrhage must be accurately determined, not guessed at. It is necessary first of all to palpate the uterus; secondly, to inspect the genitalia; and thirdly, to examine the placenta itself. There is a strong tendency to forget asepsis in the immediate urgency of post-partum hæmorrhage.

Having decided that the hæmorrhage is neither from the cervix nor from the vaginal canal, what are the indications which point to the fact that the patient is losing an excess of blood; obvious or concealed?

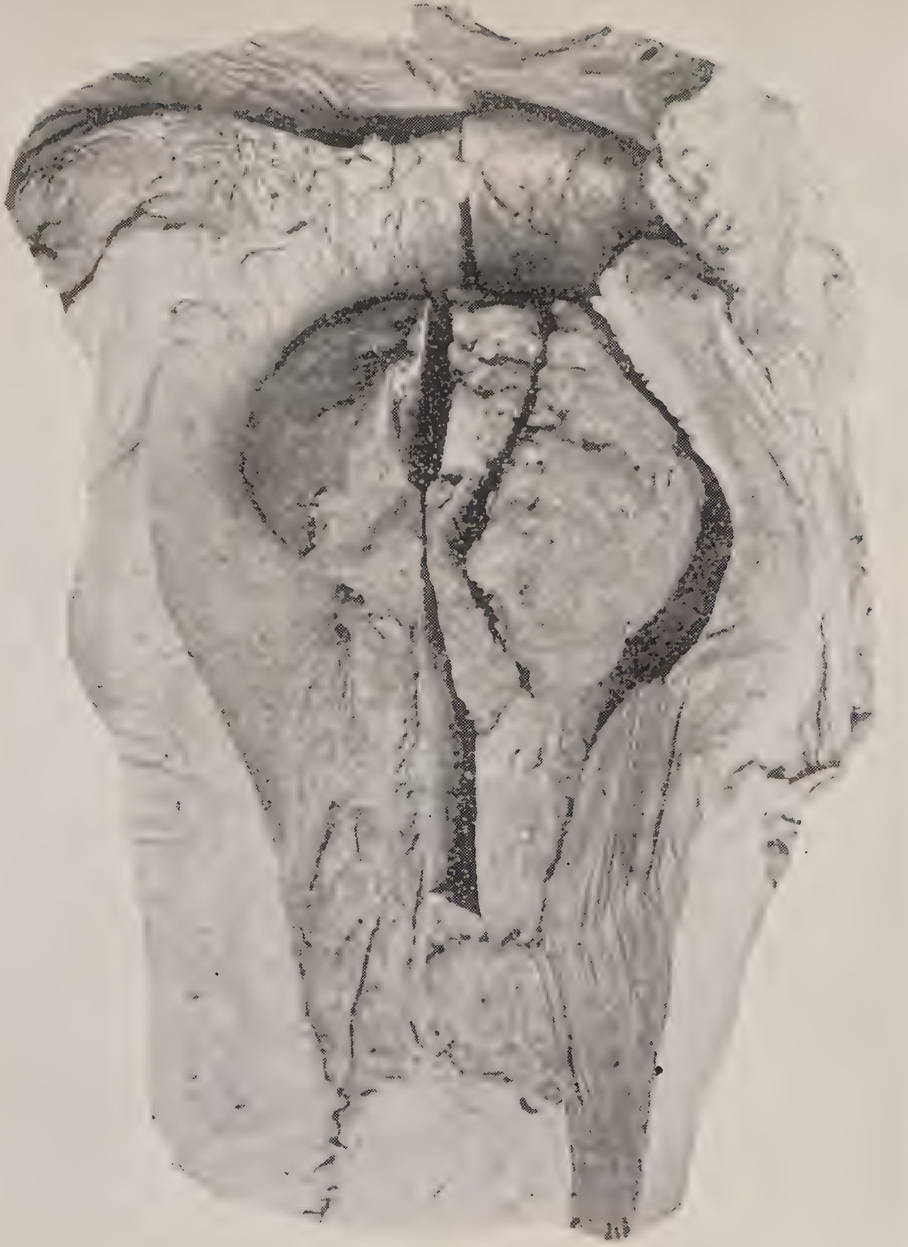
Firstly—The loss may be obvious by persistent flow; or,

Secondly—By violent gushes, and

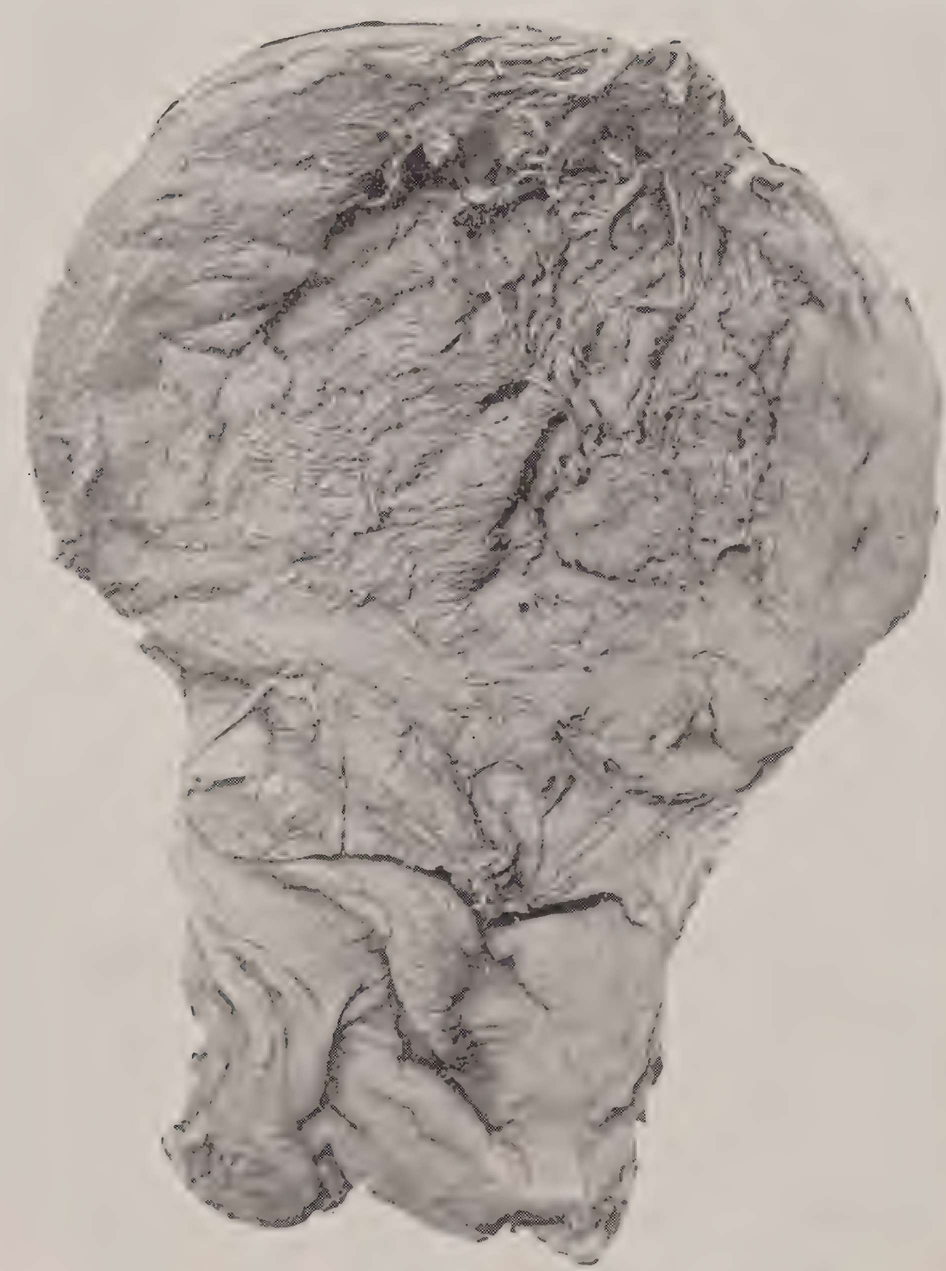
Thirdly—Collapse from concealed hæmorrhage, which is the most dangerous form.



Adherent Placenta—Death from Hæmorrhage.



Adherent Placenta—Death from Hæmorrhage.



Placental Site Torn Away—Conviction.

Treatment.

For the first, the uterus, if dilated, must be made to contract—

Firstly—By external massage of uterus.

Secondly—By administration of 15 m. of the solution of pituitary extract, hypodermically given into the buttock, or by hypodermic injection of ergotin (either can be repeated), &c.

Thirdly—The injection of water reduced from boiling point to 118° F., through a sterile glass tube, into the uterine cavity and vagina.

Fourthly—Bi-manual compression of the uterus.

Fifthly—If that does not suffice, by packing the uterine cavity with sterile gauze, soaked in adrenalin 1–10,000.

Sixthly—Application of Momberg's method—tourniquet around abdomen.

Then we proceed to—

Seventhly—Submammary transfusion with pints of normal saline at 100° F. (B.W. & Co. tabloids).

Eighthly—By the continuous saline instillation by Moynihan's or Ramsay's apparatus. The latter I always use.

All of these, rather than to introduce the hand, gloved or not, sterile (?) into the uterine cavity, and attempting to seek for and remove adherent or partially adherent pieces of placenta, and so producing profound shock and increasing the very collapse which we desire to avoid. In other words, in severe post-partum hæmorrhage, when introducing the hand into the uterus, we add a surgical operation and shock to a patient who is already collapsed.

For persistent hæmorrhage, having ascertained that the uterus is contracted, then careful ocular examination of the parturient canal must be made by means of Sims' speculum, directed especially to the regions of the perineum, the vulvæ, the clitoris, the cervical canal, and, of course, any vessel seen to bleed must be closed with sterile catgut or silkworm-gut. A practical point is that the bladder must be emptied before any manipulation during the third stage or the treatment afterwards.

Conditions liable to be overlooked are laceration of the cervix and laceration of the vaginal tissue.

Every practitioner attending an obstetric case should carry a Sims' speculum, depressor, tenaculum, needles, and holder, sterile silkworm-gut, and gauze, and while using one pair of sterile gloves for the confinement, should have another pair ready for use during the third stage, and never be without three yards of rubber tubing and some ampules of pituitary extract and ergotin.

(N.B.—Not to anticipate post-partum hæmorrhage, or needlessly to hurry the third stage, is simply to mismanage the case.)

Supposing that the third stage has been carefully completed, the placenta having been expelled naturally, or expressed by a carefully carried-out Dublin method, after waiting half an hour or one hour, and yet severe hæmorrhage takes place. Where does that hæmorrhage usually come from? In my opinion, certainly *not* from that area to which a piece of placenta may be adherent. Obviously, if a piece of placenta is adherent to a given area, the vessels in that area and under that piece of placenta are occluded by it. The cavity of the uterus itself may be almost as great as if the whole placenta were within it. In a case where such post-partum hæmorrhage occurs as to produce profound pallor and almost imperceptible pulse, coldness of body and extremities and air-hunger, I would earnestly advocate that under such circumstances it were wiser, safer, and fraught with better results, immediate and remote, to induce firm uterine contraction, but not to attempt to pass the hand into the uterus and seek for a piece of placenta which may or may not be there.

Does a piece of retained placenta, adherent or not, account for uterine atony? I believe *not*.

It is quite true that the insertion of the hand into the uterus might possibly cause the uterus to contract under such stimulation—an utterly fagged horse may make an additional effort under the whip—but it were in my opinion wiser to avoid the great risks of shock and sepsis and employ the methods I have already indicated. This refers especially to cases where the practitioner is probably miles away from competent help; where, in the emergency, he has neither time nor opportunity to thoroughly sterilise his instruments or his hands. I hold that his first duty should be to ascertain that the hæmorrhage does not come from any traumatism, and, having assured himself on that point, I am convinced, by experience, that hæmorrhage will not recur if a fagged uterus can be made to completely contract and be kept contracted, even if pieces of the placenta remain. Should he feel certain that "something has remained in the uterus," it were wiser to leave it until by signs and symptoms it gave evidence of its presence, or until he could make arrangements for competent assistance from a brother practitioner in administering an anæsthetic and securing competent nursing. Then the patient, having conserved her strength and even regained much that she had lost, is in a better condition to have the uterus cleaned by the surgeon's sterilised gloved finger, or by a sterile blunt curette if there be any evidence of sepsis. Such operations can be delayed for two or even five days.

Supposing a piece of placenta has been left in utero, either due to faulty management of the third stage or by inflammatory adhesion, why worry about it at the time of emergency? Think of the right cause of the hæmorrhage, viz., the loss of proper functioning of the uterus, and attend to that.

If, two or three days later, there be any doubt, it will be found that Schroeder's sign is absolutely reliable, viz., "The os internum remains patulous as long as any piece of placenta or membranes are in the uterine cavity." This sign, however, may be vitiated if ergot has been given continuously. Otherwise, I never knew it to fail, and I have demonstrated its truth frequently during consultations.

I most strongly hold that in profound collapse due to post-partum hæmorrhage, it is the height of folly to add to the condition of shock and collapse by attempting then and there (with or without anæsthesia) to enter the uterus to forcibly detach that which, in my opinion, is not the cause of the hæmorrhage.

Arnold Lee, of Manchester, "Monograph on Puerperal Infections" (page 248), says that "Manual removal of placenta is perhaps the most dangerous of all obstetric operations. If the third stage is conducted properly it should be very rarely required. In the absence of any urgent indications, no attempt should be made to extract the placenta for at least one hour after the birth of the child."

Ahlfeld and Olshausen and others say that in hospital practice the manual removal of the placenta is required in less than 1 per cent.

In an experience of some 7,000 obstetric cases, of which nearly 1,800 have been in private practice, I have learned to anticipate post-partum hæmorrhage, if I am dealing with patients who are the subject of—

1. Syphilis or Bright's disease.
2. Goitre.
3. Myxœdema.
4. Who have had a prolonged dystocia in the second stage.
5. Whose chloroform anæsthesia has extended over an hour and a half, even if the anæsthetic be carefully given, or after scopolamine-morphine narcosis.
6. More certain still, I should anticipate post-partum hæmorrhage if chloroform (not ether) had been given in over-dosage, no matter whether the actual delivery by instrument or turning by hand had been short or prolonged.
7. Cases in primi-gravidæ above 40 years, with some debilitating disease, such as malaria, or true tropical dysentery.
8. Where the onset of labour has been dreaded, and where the emotions came into constant play for a long time before accouchement, or where some nerve storm had greatly shaken the patient before the confinement.
9. In a woman who has borne children in rapid succession.
10. In hour-glass contraction of uterus.
11. In a case of placenta prævia.

The only death I have had in private practice was that of a case of placenta prævia I saw in consultation. She died from hæmorrhage and sepsis.

I have learned, as indeed all of us must have learned, the extreme folly of congratulating ourselves directly the child is born, and feeling that we are entitled to a rest after labour, whereas our bounden duty is immediately to ascertain—

Firstly—That the uterus is completely contracted, and remains so. (Leave the infant to the nurse, and direct nurse how to resuscitate if that be necessary.)

Secondly—That traumation of the maternal parts is not followed by hæmorrhage.

Thirdly—And never to hurry the third stage unless under some very exceptional circumstances.

I generally wait half an hour after pulsation of the cord has ceased before I attempt to express the placenta.

A completely adherent placenta is not associated with post-partum hæmorrhage if the uterus be firmly contracted.

As surely as the careless administration of the anæsthetic causes the uterus to delay its contraction, so just as surely, perhaps more so, does a delay in using instruments carefully and at the right time cause the woman to become so exhausted that her uterus cannot contract completely and efficiently.

I am a strong advocate for the immediate and careful application of forceps in cases where experience tells me that without their aid the uterus would be *unable* to expel the child, and I would strongly insist on the *cautious* use of the anæsthetic during the second stage. Still more would I most earnestly state that I have never regretted paying constant attention to the uterus directly the child is born, and particularly to the right way to perform the Dublin method of expressing the placenta, *i.e.*, Credé's. Of course, if violent hæmorrhage occurs immediately following parturition, due to an obviously relaxed uterus, and if the placenta were still in the cavity of the uterus, I would try and express it, and keep the uterus contracted, and immediately inject pituitary extract into the buttock. That failing to arrest the bleeding, I would remove placenta in usual way.

For later cases, after removal of placenta, Franz Weber, of Munich, in Doderlin's Clinic, considers tamponade to be the very best treatment, performed in a proper manner with sterile gauze under every aseptic precaution, it being especially important to pack the whole cavity of the uterus, and the vagina should also be packed. He allows it to remain for six hours at the most, when the uterus will be found firmly contracted, and hæmorrhage does not recur, because—

Firstly—The pack acts as a foreign body and the uterus tries to rid itself of the same.

Secondly—It acts by mechanical pressure upon the lumina of the bleeding vessels.

Thirdly—The blood, coming into contact with the gauze, clots more readily, and thrombosis in the vessels occurs.

On the other hand, Ahlfeld and Veit oppose packing. Veit says he has not seen a single case of post-partum hæmorrhage which would induce him to pack on account of hæmorrhage. Personally, I dreaded the risk of sepsis wherever I have used the uterine tamponade. I seldom use the tamponade now because it distends the uterus rather than making it contract.

At the Basle Women's Hospital, out of 10,000 confinements seven died from post-partum hæmorrhage, of which six had been tamponaded.

Weber states that the use of Momberg's method in many clinics is so favourable that it must be considered a dependable one in serious cases of uterine atony, and as such is to be recommended to the general practitioner.

Momberg's method consists in passing a stout rubber tube several times round the body between the pelvis and the lower costal margin, and tightening it until pulsation in the femoral arteries ceases. The patient frequently requires hypodermic injection of morphia. The tube is about the thickness of a thumb, and 2½ to 3 yards in length. It is placed just above the iliac crest, and is slowly drawn tight by two people. When femoral pulsation ceases, the tube is fixed with a pair of forceps. It is more easily adjusted if it be oiled first, and as a rule, it is not necessary to wind it round the body more than twice. When pressure is made, the coils of the intestines slip up towards the thorax, but if any of them be compressed, they can remain so for a considerable length of time without sustaining injury. The ureters also escape damage. The tube is kept tight for ten to thirty minutes, and it is slowly relaxed, the lower limbs being well bandaged first—if hæmorrhage occurs it is readily readjusted. Out of thirty cases reported, twenty-eight have been successful. Of these, eight were due to atony of the uterus, twelve to retained placenta, and two to laceration of the cervix. This method gives the obstetrician time for disinfecting his hands and the parts before further manipulation. Patients tolerate the treatment well, though collapse may follow the removal of the ligature, the blood rushing into the lower limbs and producing anæmia of the brain or heart. This may be obviated by the Trendelenberg position, or by tilting the bed. Where other methods fail, this one may be the means of saving life. (*British Medical Journal*, 1st April, 1911.)

Baudeloque's method of digital compression of the aorta at level of fourth lumbar vertebra is difficult and inconvenient, and is not always effective, since the ovarian arteries, which branch off just below the bifurcation of the iliac, carry blood through the uterus, and a flow through them is not checked by Baudeloque's method—and the inferior vena cava is compressed at the same time, and that is undesirable.

The too rapid delivery of the fœtus may empty the uterus before it has time to follow the child.

I have had five patients place themselves in my hands for their confinements, each stating that her own birth had cost the life of the mother from post-partum hæmorrhage. It is a point of some significance that in each case the doctor had left the bedside congratulating himself and the father on the safe arrival of the child, and in each case within an hour sudden death occurred from post-partum hæmorrhage.

A woman, dreading that her life is in danger from post-partum hæmorrhage, feels strengthened by the mere presence of the doctor she trusts in the house, and there is no question but that such confidence assists a woman in every possible way, and, I believe, saves many a life. Fear, anxiety, deep or prolonged anæsthesia, all tend to loss of control of the muscular centres and power, and so to the relaxation of the uterus.

None of us with a large obstetric experience could fail to be impressed by the obvious practical knowledge of the subject of post-partum hæmorrhage shown by Jellett in the latest edition of his text-book. And if you wish to have the use of pituitary extract placed before you concisely, I commend to your notice the paper by Dr. Jervois Aarons in the *Lancet* of 24th December, 1910, being the paper he read at the Fifth International Congress of Obstetrics held last year in St. Petersburg.

In the short time at my disposal I would not bore you by the recital of individual cases, and I feel that I have not done justice to this most important subject. If I have brought nothing new to your notice, I trust I have at least shown that whereas nowadays the science of obstetrics has advanced so that we can successfully fight sepsis, yet we are liable to forget the art of midwifery, which teaches the practice of extreme care and patience in guarding a woman during the whole period of her gestation and parturition.

HÆMORRHAGES OF PREGNANCY.

DISCUSSION.

Dr. J. A. G. HAMILTON: The most important treatment in dealing with hæmorrhage is efficient plugging of vagina in placenta prævia, and uterus and vagina in post-partum. The plugging is only efficient if carried out thoroughly, with gauze in strips not too wide, and saturated with a solution of adrenalin; or, in my opinion, a solution of iodine is more efficient. Hot saline in cavity of uterus is most efficient, in my opinion; but it must be used very hot, and in using it hot it is well to protect the vagina, as the hot water coming over the speculum may cause a burning of vagina. It has been suggested by some American writers that cases of placenta prævia should be treated by Cæsarean section. It does not appear to me that there are many cases which call for this treatment. I would like to emphasise the importance of the use of pituitary extract in post-partum hæmorrhage as being most efficient, and also the use of some reliable preparation of ergot.

Dr. R. TATE SUTHERLAND: With regard to the causation of concealed ante-partum hæmorrhage, I want to draw brief attention to two factors. I am dealing with those cases in which external bleeding is not noticed:—

First—That the intermittent uterine contractions are not limited to the period of labour, but occur throughout pregnancy, and, in fact, throughout functional life. It is these contractions that find out the weak spot in the placental cohesion.

Second—That the condition described as decidual endometritis—the product in many cases of syphilis—supplies this weak spot, either from alterations in the endometrium or from degeneration of the ovum.

But to pass to more practical suggestions—it seems a paradox to say, that in concealed ante-partum hæmorrhage the loss of blood is not the most dangerous element.

Such, however, is the fact. During labour, the uterine muscles, as a whole, are not stretched. The *lower* uterine segment is, of course, distended.

Concealed hæmorrhage rapidly puts on tension the muscularis of the *upper segment*, and thus produces the dangerous shock so often seen, with distension of circular fibres, whether of uterus, cervix, anus, or bowel.

Which of the two conditions—shock or hæmorrhage—predominates, must be determined; for treatment differs widely with diagnosis.

The blueness and apathy of the one contrasts with the pallor and alertness of the other, and should in unmixed cases leave us little in doubt.

If shock, buck up beforehand—remember that the removal of pressure of uterine contents relaxes the intra-abdominal pressure and allows veins to dilate and take up more of the already scant supply of blood.

This is the cause of death immediately after delivery in these cases.

Dr. H. OSBURN COWEN: With reference to the occurrence of accidental hæmorrhage, Dr. Cowen said he was pleased to hear Dr. Cameron's reference to Dr. Davies Colley's paper on the association of toxæmia with the hæmorrhage of pregnancy. He had thought this one of the most careful pieces of work in obstetrics which had come under his notice for some years. In the second case mentioned by Dr. Cameron he would suggest that when accidental hæmorrhage occurred, the uterus should have been emptied without delay. Hermann had pointed out that the presence of even a small clot between the placenta and the uterus acts as a foreign body, and leads to still further separation and more serious and perhaps appalling hæmorrhage.

Referring to Dr. Dunbar Hooper's careful statement of the causes and treatment of post-partum hæmorrhage, he thought the suggestion that portions of adherent placenta should be left was a dangerous one. There might be cases where the exhausted patient could stand no more, but as a general principle he was entirely opposed to leaving portions of adherent placenta. It seemed to him as unsurgical as the expectant treatment of ectopic gestation, in which the diagnosis of intra-peritoneal rupture has been definitely established. As to causation, he was of opinion that the too early and too frequent use of the forceps was the cause of much of our post-partum hæmorrhage. It was significant that as Dr. Hooper had pointed out, post-partum hæmorrhage was unknown among aboriginal races and the anthropoid apes—those were cases which were spared what is frequently the outrage of forceps delivery.

He had not found the manual removal of the placenta attended with serious risk. He had never regretted it, but had often regretted the persistence in forcible expression of the placenta. In cases of hour-glass contraction, the uterus should be excited to contraction at the time of manual removal of the placenta.

Dr. T. G. WILSON said that anyone who had had the advantage of seeing the treatment of ante-partum hæmorrhage by plugging of the vagina as practised at the Rotunda Hospital in Dublin, could not doubt the efficiency of this treatment, but he thought that often the method was not done properly. He had had cases sent into hospital who were supposed to be plugged, but when one came to remove the plugging it was found to be quite inefficient. The plugging must be done thoroughly, and it is remarkable how much such a vagina will hold, and, combined with a perineal pad and tight abdominal binder, he thought this method was the method of choice when there was no or little dilatation of the cervix. He had had experience of two cases of accidental hæmorrhage of severe degree, in elderly primiparæ with rigid undilatable cervices, and should he meet such another case would be inclined to advise vaginal Cæsarean section if the surroundings were suitable. In ante-partum hæmorrhage the life of the mother and of the child are inimical to each other, but in the condition mentioned above, he thought that this was probably the one condition where a Cæsarean section would give both the best chance.

With regard to severe post-partum hæmorrhage, he thought the introduction of the hand into the uterus and combined pressure gave the best chance. He was glad to hear Dr. Dunbar Hooper advise the carrying of pituitary extract in the midwifery bag, as he had seen several cases where this drug had been most successful in causing the uterus to contract. In a case of Cæsarean section he had last year, where the uterus was very soft and flabby, after the removal of the placenta, the uterus had contracted and stood up like a cricket ball, immediately following the injection of about 15 minims of 1-1,000 adrenalin solution into several places of the uterus itself. The immediate response to the injections in this case had been most remarkable, and Dr. Cudmore, who was assisting him, and Dr. J. C. Verco, who was present, both remarked on the almost immediate difference in the condition of the uterus. The reports published from the use of Momberg's tube had been satisfactory, and the speaker had hoped to hear if any of those present had used this method, as he himself had had no practical experience with this appliance.

Dr. CHENHALL: Treatment of hæmorrhage has been so fully and ably discussed that I have little to add. The only case of hour-glass contraction of the uterus I have ever seen occurred in the practice of a colleague who had administered ergot before completion of the third stage of labour, and then asked me to relieve him whilst he attended another case.

Ultimately, after administration of chloroform, I was able to remove an adherent placenta.

I wish, however, to address myself to the causes of ante- and post-partum hæmorrhages, and the great economic loss dependent upon occurrence of abortion, the most fruitful source of hæmorrhages.

Since the economic loss resulting from so many abortions is enormous, and the suffering, both present and deferred, which arises therefrom is great, it becomes our duty to seek causes and reduce them as far as possible. These causes are almost all completely avoidable.

Apart from accidental causes, both physical and mental, due to injury or shock, which, with ordinary care on the part of pregnant women and those responsible for their protection during pregnancy, may be avoided; and apart from serious constitutional ill-health and infectious fevers, I believe abortions are due to four important causes:—Deliberate induction, syphilis, endometritis, and lacerated cervix uteri, usually accompanied by some displacement. Moreover, I believe endometritis, with lacerated cervix and accompanying subinvolution of the uterus, to be the all-essential cause of placenta prævia, hence indirectly responsible for all the hæmorrhages arising therefrom.

Hence our chief aims should be directed against these predisposing conditions which are the main causes of the hæmorrhages of pregnancy.

Legal enactments should deal much more effectually with deliberate induction, if education and instruction continue to fail.

Coition should be interdicted during occurrence of endometritis, and the disease dealt with by careful treatment.

Syphilis should be brought into correct focus for the public eye, so that its existence may be guarded against in marriage.

Possibly here salvarsan, by early administration, may save disaster.

Displacements of the uterus should be corrected and recurrence guarded against by normal action of the bowels, relief from superincumbent weight of clothing by carrying it chiefly supported from the shoulders, by cure of endometritis on accepted medical and surgical lines, and by careful repair of a lacerated cervix. Here, incidentally, I would state my belief that most severe cases of lacerated cervix are due to improper use of forceps on the undilated cervix and ill-judged force in delivery of the child—hence are almost completely preventable.

I have drawn attention to this aspect of the discussion because it has not been dealt with by others who speak regarding the diagnosis and treatment of abortion.

Experience has taught me that in diagnosis one must guard against error in the rare contingency that pregnancy may exist in one horn of a bicornuate uterus and menstrual secretion escape from the other.

More important still is it to remember and determine early whether the case is one of threatened abortion or a case of extra-uterine pregnancy, since early recognition of the latter may lead to successful treatment before catastrophe.

Dr. F. J. Newman (Geelong) thought that sufficient reference had not been made to the easiest and most effectual method of stopping post-partum hæmorrhage by grasping the uterus and turning it forward and pressing the lower segment against the pubis.

Of course, anything done internally should be under strictly aseptic conditions.

Before any manual procedure, necessarily the injection of hot sterilised water or saline into the cavity of the uterus is essential.

With reference to the statement of Dr. Cowen that the constant use of forceps was almost criminal, I can only say I use forceps constantly, believing firmly that although Nature's methods in nature are good, still Nature has sometimes to be helped.

Constipated people do not find it pleasant to wait for Nature's methods, and most (even scientific) opinions agree that constipated people feel fairly relieved when Nature is helped a little.

As to waiting an hour and two hours (as some speakers recommended) until the placenta is expelled, and not previously trying to help in its removal, most practitioners could testify that they rarely wait more than twenty or thirty minutes for the natural expulsion of the placenta, and then they use Credé's method of expulsion of the placenta.

It is absolutely of the greatest importance that practitioners should understand that all obstetric work should be under the same strictly aseptic conditions as the most delicate surgical work. One will have very little trouble after removing a placenta if you are aseptic in your methods.

Dr. Crowther, of Tasmania, as a general practitioner, with a now extensive midwifery practice, challenged four statements, and it was on behalf of the younger men who, *alone, far from available assistance, had to decide* and act, that he did so:—

1. The use of adrenal gauze to pack the uterus with in post-partum hæmorrhage.

2. The leaving of any (known portion) of afterbirth or other substance in the uterus for any time whatever.
3. The caution (so strongly put) to be so *very* careful not to use the hand inside the uterus.
4. The waiting time named (was more than necessary) after birth for placenta to come away. What was possible in a hospital with all available help was next to impracticable under the usual surroundings of general practitioners, especially in the country.

His plan was to follow the afterbirth and never let his hand off the abdomen until *all was right.* If in one hour it had not come away, he made it the universal rule (under the strictest aseptic care) to reintroduce his hand and clear out the uterus (never with gloves), and he had never lost a case. This rule applied to *all* or a *bit* of afterbirth. The one hand in and the other outside the uterus was worth all the other methods in these post-partum dangers *when properly used*, and *could contract* the bleeding. As to anticipating loss of blood at full time:—Stick to the patient. Plug and re-plug, and as soon as you can get the womb sufficiently dilated to introduce the hand, get down a foot and your danger is over. I have saved a life by using a woollen neck comforter to plug with. I was far from any help when practising in Lincolnshire, in England, and with this I *plugged*, *waited*, *turned*, *and delivered*, and woman and child lived.

I have never had *a death* or seen a bad result from what I have stated.

Dr. TAYLOR DOWNIE (Melbourne) advocated the use of ergot by the mouth at the end of the second stage of labour, unless where immediate action was required, then the placenta should be removed, and either a prepartion of ergot or pituitary extract given hypodermically.

He also drew attention to the value of the pulse-rate, in deciding whether the patient was losing too much, and thereby coming under the head of post-partum hæmorrhage.

Dr. BARRINGTON suggested that in regard to ante-partum hæmorrhage there was a definite field for vaginal Cæsarean section when the child was small, the cervix rigid, and urgent delivery was needed. He thought the remark of Dr. Cameron—" very little post-partum hæmorrhage turns the scale "—full of wisdom. For this reason rapid completion of the third stage and plugging of the uterus and vagina was often imperative, and this could be efficiently done as the patient was usually under an anæsthetic. This stopped the continuous venous post-partum trickle, which might be regarded as the last straw as determining the fatal issue in some cases of placenta prævia. One of Dührrsen's tins of sterilised gauze could be relied upon for this purpose, and he always carried one in his bag. Post-partum hæmorrhage was very rare if there was no hurried third stage. In a previous paper he had advocated the use of the gloved hand in manually extracting an adherent placenta. By avoiding septic contact and using gloves for septic cases, the bare hand could do the work more speedily and more efficiently. He understood Dr. Hooper to advocate that in profound collapse from post-partum hæmorrhage it was not good practice to endeavour to remove small pieces and tags of placenta, and with this advice he entirely agreed. Such remnants could be removed with greater safety when the collapse had passed off. He strongly advocated the use of pituitary extract intra-muscularly in place of ergotin as a routine after the completion of the third stage, as its action was rapid and efficient. He regarded the use of Momberg's method in post-partum hæmorrhage as a means of torture, and bimanual compression of the uterus far preferable, as it was surely much better to efficiently compress a bleeding organ than endeavour to compress the vessels leading to that organ. He restricted the use of scopolamine and morphia to the first stage in primiparæ when they were getting tired, as he regarded it dangerous in parous patients, owing to the fact that the child was often born blue, and great difficulty was experienced in getting it to breathe properly.

Drs. S. H. MACCULLOCH (Sydney), HONE (Adelaide), RALPH WORRALL (Sydney), and FOREMAN (Sydney), also took part in the discussion.

After the Chairman reviewed the discussion, Drs. Cameron and Dunbar Hooper replied.

Dr. DUNBAR HOOPER (in reply) explained definitely that in cases of severe post-partum hæmorrhage associated with flabby uterine contraction, and the entire placenta remaining in the uterus—he would immediately try to make the uterus contract and eject the placenta; failing that, immediately insert a clean hand and remove placenta in usual manner, followed by repetition of hypodermic injection of pituitary extract and injection of sterile hot water. But in cases of post-partum hæmorrhage associated with the suspicion or certainty that a piece of placenta remained *in utero*, he would not bother about it, but proceed at once to make the uterus contract, as already laid down, heal the collapse, and a few hours later—or even a day or two later—when skilled assistance may be procured, then remove under ether and with aseptic technique. But Dr. Dunbar Hooper knew perfectly well that he was attacking established authority. If, however, authority can never be attacked, progress in science will cease.

SURGICAL TREATMENT OF UTERINE RETRO-DISPLACEMENTS WITH RESULTS OF OPERATION.

By A. NORMAN MCARTHUR, M.B., B.S. (Melb.), M.R.C.S. (Eng.), L.R.C.P. (Lond.).

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Methods of Replacing the Uterus.

1. Digital manipulation for retroflexed uterus can never be anything else but temporary.
2. Intra-vaginal massage, as adopted by the French, is malicious.

3. Pessaries are unscientific, and do harm by pressure, destroying or causing atrophy of certain groups of uterine ligaments. The round pessary might be better used for parlour quoits, the various others as drawing-room puzzles.

As yet, the only satisfactory methods are surgical. But there are many disappointments in all surgical methods. If gynæcological surgeons doubt this statement, I recommend them to do as I did—call in and examine all hospital patients for the last six years, and note the after results of operation. General hospital patients, because the operation is done under the best possible conditions, and convalescence, outside the hospital, usually under the worst. This test is a severe one. It is an excellent method for a young gynæcologist to reduce threatened "megalo-cephalos."

Before such an august body I give my experiences, not in the form of statistics, and I do not pretend to instruct, but hope to learn. My failures have taught me much, and though I am quoting you principally a few failures, I beg of you to believe that a large majority are successes which will not be referred to. To accept this statement will appease my vanity.

I think that in retroposition of a uterus there are certain factors that must be considered.

The uterus has a definite form and shape—pyriform, and under normal conditions is capable of maintaining that shape. The acme of normal anteflexion is at the junction of cervix and body. The lower segment of the uterus is embedded in the pelvic floor, while its upper end is *free* and movable and rests upon the superior surface of the bladder.

The normal angle of flexion of the uterus resting upon the bladder varies continually. Receiving little support from its peritoneal and other attachments, is influenced by the changing condition of the bladder.

The anterior surface of the uterus, following the changes of the upper vesical wall upon which it lies, very seldom contains intestinal coils.

The uterus is connected with the bladder, ovaries, abdominal wall, to the floor of the pelvis and the vagina. Most of these attachments or so called ligaments have little influence in supporting the uterus, but owing to the intimate connection of the cervix with the vagina, and then with the pelvic floor and with the sacrum by fibro-muscular bands, the lower segment enjoys a relatively fixed position; the body, on the contrary, being freely movable.

From this clinical aspect I wish to show that of the four sets of so-called ligaments the round ligament is the only one that has any defined shape or form. Its uterine origin is less than half an inch below and in front of the uterine ostium of the tube; it passes laterally outwards, and not till after it passes the true pelvis does it take a slightly forward and upward direction. I wish also to remind you that the inguinal end is weaker than the uterine end. In doing a panhysterectomy, the upper part of the broad ligament, so-called, is distinctly shown to be only two layers of peritoneum, in which a few blood vessels are loosely held in position by weak filaments of connective tissue, but no use as supports to uterus.

It is not till we get to the cervix, when the uterine artery is found to lie upon a dense fibrous mass (but indefinitely outlined), the first firm material worthy of being called a ligament that is felt to fill the bite of the scissors. It is attached to the lateral wall of the cervix and upper wall of vagina, spreads out into fan shape, passes outwards and slightly backwards to be united to pelvic fascia. Mackenrodt calls this the ligamentum transversalis colli, and when cut at once releases the cervix from an apparent fixation. In much the same way the uterosacral ligaments can be defined, but the fibro-muscular tissue is not so dense, and the peritoneal fold is closer down upon the actual ligament. These ligaments are attached to the cervix posteriorly and towards outer edge. They fan out to an attachment to the sacrum on each side of the rectum and to the rectum itself. The round ligaments are merely guide ropes to the uterus, and are in no way suspensory.

Causes of Retroposition.

Congenital loss of resilience of the whole of the uterine structure, permitting of either complete retroflexion or anteflexion, according to circumstances.

Congenital weakness of the supporting ligaments, especially uterosacral, which, when lax, will permit of rotation around the axis formed by the attachment of ligamenti transversales colli.

Traumatic displacement of uterus in nullipara can occur *acutely*, when jumping from a height on to the heels, as from a vehicle; or falling in the sitting posture, as in skating; or on the stairs during descent.

Or, *chronically produced*, as by constantly lifting heavy weights, prolonged standing, or very commonly in girls who habitually use very heavy treadle sewing machines.

In multipara the same condition is produced by the trauma of parturition. You can readily see that the brunt of expulsive labour pains will act adversely more upon the uterosacral than upon any other ligaments because of the marked curve (with concavity anteriorly) of the pelvic plane. But without destruction of these ligaments there are other causes—retro-uterine peritonitis, contracting adhesions, increased weight of uterus, chronic constipation and resultant straining, soft mobile condition of uterine walls.

Various surgical methods have been devised to keep and fix a retroposed uterus in position. To my mind the stretching or destruction of the uterosacral ligament having such a great influence upon these retrodeviations to direct to them one's remedial efforts. But any effort to shorten or fold upon itself the uterosacral or broad ligament is ineffective, as the structure is so ill-defined in outline, though it can be readily felt when put in the stretch or gripped in the bite of the scissors. So long as the sutures last and do not cut through, efforts at shortening uterosacral ligaments are well enough, but later, when sutures become absorbed or cut through, the ligament relaxes worse than before, and lately I have quite neglected making further use of them *per se*.

One of the oldest methods, and one that has had marked fluctuations of popularity, is external shortening of round ligaments (after Alexander Adams' suggestion), and under all conditions will give many very disappointing results—the reason being obvious. The round ligament is merely a guiding rope, and a normal uterus can be placed in a position of retroflection without any tension at all on the round ligaments. The uterus's own resilience will bring it back to normal position. Moreover, as I have demonstrated frequently to the students in the operating theatre, the round ligaments have almost a purely lateral course, with just a very small anterior direction. It becomes, then, perfectly obvious, when the pull of a shortened ligament is nearly all in a lateral direction, there is a tremendous loss of power in these ligaments attempting to prevent the uterus taking a backward direction.

The limitations for such operation.—In nullipara—uterus not larger than normal; one that can be easily replaced without any snap back; and one in which there has been no previous history of pelvic inflammation to cause adhesions.

In multipara the limitations for this operation are even more marked. After parturition the retro-deviation so often has completely returned that I hardly ever do the operation in these cases.

The Incision should be through the skin only. Fat opened to intercolumnar fascia by forceps. Never open the external ring. There is no advantage to be gained, and no sutured external ring will be as strong as a normal one. Search in the hollow of Poupart's ligament for round ligament; and branches of the ovarian artery which are minute and markedly convoluted, like the spermatic vessels in the male, are the best guides for the ligament. Do not pull the ligament out too forcibly to overstretch it. Do not push the finger into the abdomen to break down adhesions. It is inviting post-operative hernia to occur. If there are adhesions they cannot so readily be dealt with and the mid line is the way in which to deal with such a condition, using any of the methods of shortening which I am about to criticise. After all, external shortening of the round ligaments is rightly an extra-peritoneal operation, and should never be anything else.

There is a method of internal shortening, Montgomery's I think, closely allied to an external shortening; more difficult to perform, but a little better in results. To do it, incise the peritoneum over the middle of the round ligament, carry a piece of silk round round ligament at this incision so as not to include the peritoneum. Thread both ends of silk through the eye of a bold blunt curved needle, and pass needle through internal ring, and inwards over muscle, then through fascia to within an inch of incision, and there fix with chromicised sutures. It is claimed that this method is good because the middle strong part of the ligament is used; that no peritoneum is dragged through with it, and that, having passed through the external ring and at once taking an inward direction, a slightly more direct anterior pull is given to uterus. I think I have only done about eight cases in this way, uncomplicated, simple cases, but all did well. But I see little in it to recommend it above external shortening.

The most common method adopted to surgically replace a retroverted uterus is *ventro-suspension*, as originated or elaborated by Howard Kelly. The ease and rapidity with which it can be done is a great attraction. But, alas, I have discovered that we have absolutely no control over the degree of adhesions that will form between visceral and parietal peritoneum. I have had many opportunities, for other surgical conditions, to open abdomens of those who have had the operation of ventro-suspension performed previously. The majority showed the orthodox band of adhesions, not too much, and not too little. But, alas, a few showed a dense mass of adhesions, like a fixation; while in others, the uterus had returned to the old retroflexed position, and a remnant of adhesion in a long thread from posterior wall of fundus to abdomen. I cannot be persuaded to believe that the amount of adhesive band can be nicely adjusted to the suture that is used and extent of peritoneum that is picked up. I have seen perfect results with silk, silkworm gut, chromicised catgut, and xylolised gut—I have seen the uterus grossly adherent after the use of each. Also, I have found the uterus retroverted as badly as ever after having used each of these sutures. I have given up xylolised gut for suspension, as I am now quite certain that in some cases it does not last long enough to give a good adhesion.

The abdominal surgeon's experience is that there is no law controlling the degree of any abdominal adhesions.

There are various methods of internal shortening of round ligaments. Folding the ligament upon itself—in which the pull of the ligament is still lateral. That part of the ligament that enters the internal ring and runs outwards and forwards to the external ring is the weakest part, and is less able to bear any pull. For these two reasons this method is not good.

The same applies to all other methods in which the outer part of the ligament is not fortified. As, for instance, in *Baldy's method—" Looping the loop,"* as my students call it—in which the round ligament is grasped in the centre and is brought through the broad ligament underneath tube and utero-ovarian vessels, and the loop stitched to posterior wall of fundus. There is a little more pain on convalescence, and I have always thought that there might be some vascular congestion in the broad ligament. This was very definitely proved to be correct, as only lately I had to open an abdomen for some other surgical investigations and the " looping of the loop " was perfect, but the utero-ovarian veins had become much distended and engorged as they passed over the ligament as it pierced the broad ligament.

In a case done by this method the position of the uterus was quite good for six months, but twelve months later was completely retroverted with a marked anteflexion, indicating that the round ligament had stretched at abdominal or inguinal ends.

In order to avoid a pull on the weak abdominal end of the round ligament, *Gilliam devised a method* in which the round ligament was grasped by forceps 1 inch or 1½ inches from uterus, and drawn through the abdominal parietes about ¾ inch from the edge of the central incision of abdomen, and then stitched to deep fascia. This operation has given me the happiest results, although I have not had a sufficient number who have borne children subsequently. I think, however, that the pregnant uterus will not drag upon this very much, for I have been very much struck with the extraordinary hypertrophy of uterus from above each round ligament as compared with the development below them.

However, in Gilliam's method there are two bands for intestinal kinks to occur upon, instead of one band as in ventro-suspension. Moreover, where the peritoneum has been dragged through into the parietes a funiculus is formed which favours the probability of subsequent hernia. I cannot trace a case in which obstruction to the bowel has occurred in Gilliam's operation or in ventro-suspension, but I believe other surgeons had this experience.

To overcome these possible objections I have devised an operation which, so far, has served me well. I split the peritoneum on the anterior surface of the round ligament 1 inch from uterine end of ligament to internal ring, giving a free raw surface on greater part of round ligament. Then I attach with running suture of chromicised gut the raw surfaced ligament to the parietal peritoneum from internal ring to within 1½ inches of abdominal incision, where it is firmly attached. I hope for gross adhesions between ligament and parietal peritoneum. I claim that there is no band for intestinal kinking. There is plenty of room for intestines to pass between ligaments without any possibility of obstruction. There is no funiculus for hernia to pass into, and there is greater freedom for ligament in pregnancy to pass up with uterus than in Gilliam's method. The few cases I have done have given perfect satisfaction—one is pregnant seven and a half months, and has had no discomfort.

Since Crossen has published his book, I find he has had a similar idea. It is very much like Gilliam's operation. One great difference, he claims, is that he draws the ligament through parietes obliquely inwards, passing through peritoneum and muscle only, and not the external fascia. The portion of ligament towards internal ring he sutures by purse string suture to peritoneum, obliterating the passage between internal ring and ligament. He unites the two loops of round ligament together over the recti muscles below the external fascia.

Professor Watson, of Adelaide, some years ago showed me a transverse incision through skin and fat at the site of the pubic hairs; the other layers of the abdomen are opened in the ordinary way. I use this incision always for abdominal pelvic operations, except where there is a large mass or tumour to be dealt with. The cosmetic after result is remarkable. Frequently, no trace of a scar can be seen.

I have no faith at all in vaginal methods of replacing retroposed uteris.

To sum up the situation, I may say that the two most common methods of dealing with retro-displacements—external shortening and hysteropexy—are undoubtedly the most uncertain in results.

That some such method on the lines suggested by Gilliam, or as modified by Ferguson, gives most permanent results; and, looking at my photograph of a nine months pregnant uterus, I really do not think, considering the attendant hypertrophy and softening of round ligament, there will be much stretching during that period.

Also I would point out the extremely unsatisfactory results in attempting to deal with the suspensory ligaments of the uterus, as, for instance, the uterosacral or any part of the broad ligaments. The round ligament is the one we always come back to.

I have introduced this very commonplace, well-discussed subject, to endeavour to find out if anything new is known in the way of dealing with this gynæcological trouble.

OPERATIVE TREATMENT OF RETRO-DISPLACEMENTS OF THE UTERUS.

By J. A. G. Hamilton, B.A., M.B. (Trin. Col., Dublin), L.R.C.S.

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Like the poor, this subject seems to be always with us. A few years ago, almost every journal one took up contained some new operation for this condition, many of them rather riskful and experimental, most of them unanatomical; but nowadays I presume each surgeon has settled down to some particular operation which gives him more or less satisfaction. It is not my intention to weary you by discussing the many operations that have been introduced for this condition, only to give you my experience of a few of them.

Ventro-fixation, ventro-suspension, internal plication of the round ligaments, vesico and vaginal fixation, have all been extensively tried, and mostly discarded, either because some of them caused sudden and unexpected troubles in childbirth, or because the uterine displacements recurred altogether too frequently, both before and particularly after intercurrent pregnancy and labour.

Ventro-Suspension.

This operation was introduced by Howard Kelly as an improvement on ventro-fixation when it was found that the latter operation generally left the uterus firmly fixed against the abdominal wall, and often produced dystocia so marked as to require a Cæsarean section. No man in the past or present has done more for abdominal work than Howard Kelly, but I venture to think that had it not been for the halo his great reputation cast around the operation of ventro-suspension, it would not have stood the test of time as long as it did. As the operation is easy of performance, it was undertaken by every surgeon. Possibly the operation was not carried out on the lines laid down by Howard Kelly, and this helped to bring it into disrepute; however, I think the consensus of opinion now is that ventro-suspension is not an altogether safe procedure in women of a child-bearing age. For many years I performed this operation in cases where I considered Alexander's operation unsuitable, but never felt satisfied that it was an ideal procedure, and hailed with pleasure each new operation reported for uterine retro-deviations, many of which I tried with varying success.

The objection I have to ventro-suspension is that one never knows what is going to happen after the operation, and relapses, especially after pregnancy, were much too frequent. I have performed ventro-suspension 136 times, and have always carried out faithfully the directions as laid down by Howard Kelly. Of course I have not been able to follow all the cases after operation, but found the results were either faulty or failed to permanently relieve the retro-displacement in eighteen cases. I had occasion to re-open the abdomen in twenty-eight cases, either for recurrence of the trouble or for further adnexal trouble; in some cases the uterus was held by a firm broad band, suggestive of trouble during the expansion of the uterus during pregnancy; in others I found a long thin ligament 2 or 3 inches long, apparently taking no active part in keeping the uterus in position, and giving one the idea it might at any time cause intestinal obstruction; whilst in twelve cases the band had entirely disappeared and the uterus was lying back in its former faulty position. In most of the cases where the uterus was fixed by a strong band, either infection of the abdominal wound had occurred, or kangaroo tendon or chromic catgut had been used as a suture material; in other cases plain iodine catgut had been the suture material used.

The failure of the peritoneum to act as a reliable structure for surgical reconstruction caused careful workers to look around for some more reliable and permanent structure to utilise surgically for this common disorder. This they found in the round ligaments. These structures being composed of muscular fibre derived from the uterine walls, and really a part of the uterus, and as such undergo with it the physiological changes incidental to child-bearing, namely—evolution during gestation and involution after labour—this feature in the round ligaments supply the only possibility of a permanent cure for retro-displacements, for all fixation and bands of a peritoneal or other source have no capacity to retract after labour.

The operative measures for holding the uterus in a forward position by its round ligaments are numerous; I will, however, only refer to a few representative ones of which I have had experience.

1. Extra peritoneal shortening of the round ligaments through the inguinal canal (Alexander's operation).
2. Drawing the round ligaments through an opening in the broad ligaments on each side and fastening them at the back of the uterus (Baldy's operation).
3. Transplantation of the round ligament into the abdominal wall. There are several methods of doing this—
    - (*a*) Drawing the round ligaments out through the aponeurotic wall at the internal ring and then to the median incision (Montgomery's or Mayo's operation).
    - (*b*) Out directly through the rectus muscle or its sheath (Gilliam's operation).
    - (*c*) Out directly through the rectus muscle with a suture to unite the round ligament to the abdominal wall, and thus close the opening through which an intestinal coil might slip (Gilliam-Ferguson operation).
4. Intra-abdominal plication of the round ligaments (Manns, Wylie, Dudley operation).

In giving my experience of these round ligament operations I have only looked up my records for the last five years.

Alexander's Operation.

This is undoubtedly the classical operation for retro-displacements of the uterus, but it has a limited field of usefulness; it is only indicated in those cases in which the uterine appendages are healthy, and in which the uterus can be raised to its normal position and retained there. It is claimed by some authorities that retro-displacement *per se* produces no symptoms, and that when symptoms exist they are due to complications. There can be no doubt that retro-displacement is frequently discovered by accident, and that the symptoms of uncomplicated cases are far less marked than those of complicated cases. This operation is especially indicated in virgins or nulliparous women who furnish no evidence of infection either from sepsis or gonorrhœa. Alexander's operation was introduced many years ago, but for some years fell into disuse. Of late years it has been revived, and, properly performed on selected cases, it has given excellent results. The cardinal feature of the operation is the use of blunt dissection, and the keeping the field of operation dry and free from blood. Once the tissues get stained with blood, it is difficult to distinguish the round ligaments, even when exposed.

During the last five years I have done fifty-six Alexander operations. I have only a record of two cases in which the trouble recurred. It is not always possible, even with a careful bimanual examination, corrected by a re-examination under an anæsthetic when the patient is on the operating table, to make sure that there is no disease of the appendages. I often find myself in doubt on this matter, and generally decide to open the abdomen and deal with the round ligaments from inside, and frequently find some condition in the pelvis which could only have been satisfactorily dealt with from the abdomen. Some operators, including Alexander himself, operate on all cases of retro-displacements by shortening the round ligaments in the inguinal canal, and deal with adhesions and diseased appendages through an opening in Douglas' pouch. Others deal with complications by dilating the internal ring.

Neither of these methods have ever appealed to me as likely to be effective or safe. One important complication (if it may be called so), which must not be lost sight of in deciding between an intra and extra abdominal operation, is the frequency which menstrual disturbances and pelvic pains, especially in young women with or without retro-displacement of the uterus, are caused by trouble in the appendix. A long appendix, hanging down in the pelvis and taking part in the monthly congestion, is a frequent source of pelvic pain and dysmenorrhœa.

I have had some striking results of relief from dysmenorrhœa after removal of the appendix, when dilatation, stem pessaries or various operations on the cervix had failed to give relief.

Baldy's Operation.

In this operation the round ligaments are brought through an opening in the broad ligament and fastened to the back of the uterus. I have used this operation almost entirely for the last five years for retro-displacement of the uterus in cases where I considered Alexander's operation was unsuitable, and my experience of it has been so satisfactory I thought it worth while publishing my results. It has been urged against this operation that it is unanatomical, in that the round ligaments are fastened at the back of the uterus when nature placed them in front. A similar objection would apply to all operations where the round ligaments are used to suspend the uterus, for they depend entirely for their success on suspension pure and simple, and in that the uterus is lifted out of the pelvis into the abdomen and there suspended in a position nature never intended it to be in, with lessened mobility, and the full weight of the downward drag depending alone on the integrity of the round ligaments. In contrasting this condition with Baldy's operation I cannot do better than quote his own words in his article in "The Department of Nicholas Senn Festschrift." He says:—"The uterus lifted up and sustained by the encircling round ligaments, but only temporarily and intermittently is the full force put on the round ligaments. On certain movements the fundus has a tendency to go backwards; should this occur, the intra-abdominal force would soon be exerted on the anterior surface of the fundus, and the uterus would be forced to a posterior position. In studying the mechanism of the forces at play it is soon evident that two things prevent this accident. (*a*) The sling at the back of the uterus formed by the round ligaments meets the force temporarily urging the fundus posteriorily and obstructs that action. (*b*) The force of the fundus against the encircling round ligaments causes a pull on the ligaments at their attachments to the anterior uterine wall, and the harder this pull the more the tendency to draw the top of the fundus forward and downward. In other words we have two factors acting against and neutralising the efforts of the uterus to go backwards, and it is impossible for the one to be brought into play without the other operating to aid it. Until some effort of the individual causes the situation to arise which attempts to force the uterus backward, it remains quietly forward by combination of gravity and intra-abdominal force, and no tension is put on the round ligament whatever, and it is only temporarily and intermittently called upon to exert itself, and when it does so at all it offers two points of resistance, in the obstruction posteriorily, and the pull forwards and downwards at the uterus cornu thus dividing the work put upon it; the uterus rides in a cradle, as it were, movable backwards and forwards without being able to get back beyond its centre of gravity or posterior to the direct line of axis of the pelvis. Hence the third force at play (the intra-abdominal force), is constant in its pressure on the posterior surface of the uterus, and thus trebly ensures a permanent result ; but one possible thing can spoil the operation: the complete breaking down of the suturing."

I must apologise for quoting so fully from Baldy's article, but I venture to think this operation has not been given the consideration it deserves, and that many operators have not given its mechanism a careful study.

I had performed this operation some two years before I read the article I have just quoted from, and it was not till then I fully realised the beauty of its mechanism.

Another advantage in this operation. It makes certain of permanently elevating the Fallopian tubes and ovaries. When the uterus is thrown forward by almost any other operation the ovary will usually rise in the pelvis with it, but there are a certain group of cases in which the ovarian ligaments are relatively more stretched than that of the uterus, and in spite of the fundus being brought forward the ovaries are still prolapsed. After all said and done, final results are the best test of operation. I have performed this operation 117 times. In one of my earlier cases, where the retro-displacement was accompanied by marked prolapse, I put my suture too low down on the back of the uterus, so as to raise it up as high as possible, with the result that the fundus doubled back over the encircling ligaments; but in all other cases the operation has given me a good result. Of course one cannot follow all the cases operated on, especially the hospital ones, but these cases generally turn up again for treatment when the symptoms return for which they have been operated on. This was my experience when I used to do ventro-suspension. The failures generally came to light again, and when they do not one reasonably hopes they are cured.

Technique.

When the abdomen is opened, the round ligament on one side is seized with a pair of forceps and brought back behind the uterus so as to measure the amount of shortening required, another pair of blunt forceps are pushed through a clear space in the broad ligament close to the uterus, and just under the ovarian ligament; the same is done on the other side, and the two ligaments fastened together and firmly sutured to the back of the uterus, taking care to leave no space between the fundus and round ligament. It is important in this operation to use some unabsorbable material, such as silk, or a slowly-absorbable material such as Van Horn's forty-day catgut. In one case in which I had used plain iodine catgut as a suture material, having to open the abdomen again six months later for further trouble in the ovaries, I found the left round ligament had given way completely and the uterus was only held in position by the right ligament. This is only the second opportunity I have had of seeing the result of Baldy's operation. In the second case, on opening the abdomen the uterus was found in perfect condition, snugly encircled by its round ligaments. There is one class of case in which the operator has to use special care in doing the operation. I refer to those cases where there is a varicocele of the broad ligament; these large thin-walled veins are very easily torn in putting the round ligament through the broad. Baldy's operation leaves the uterus mobile in its normal position in the pelvis, and does not fix it against the abdominal wall out of the pelvis, as most other operations for this condition do. It would be well for us to remember the lesson which Schultze has taught us for many years—that a normal uterus is a mobile uterus, and that any surgical procedure which interferes with its mobility, compromises all its functions.

Gilliam's Operation, and Its Modifications by Ferguson, Montgomery and Mayo.—I have performed Gilliam's operation six times, but abandoned it, as it leaves a space where an intestinal coil might get caught. The Gilliam-Ferguson I have performed once, but found great difficulty in suturing the round ligament to the abdominal wall. The Montgomery method I have used 12 times and the Mayo method twice.

I have three objections to this method of operating for retro-displacements:—(1) The uterus is raised out of the pelvis and suspended against the abdominal wall. (2) The weight of the uterus is entirely depending for its support on the longitudinal pull on the round ligament, and, as Baldy points out, the downward pull of the full length of the ligament has only a fraction of the chance of overstretching which obtains in the longitudinal pull, and the uterus is suspended in a horizontal position—rather than in the normal position of antiflexion, so that the intra-abdominal pressure does not come freely on the posterior surface of the uterus as it should do to preserve its forward position. (3) There is to my mind a grave risk of wounding the deep epigastric vessels in operating by any of the above methods. On one occasion I wounded the epigastric vessel in operating by the Montgomery method. I am afraid I failed to recognise the mischief in time, a large hæmatoma formed, which became infected, causing extensive suppuration, the patient eventually got an infective empyema, and only recovered after a lengthy and serious illness. In talking the matter over with Professor Watson we examined a number of bodies in the Anatomical School, and found that in several cases the course of the deep epigastric vessel altered very materially in its course, and it appeared to us, after examining these bodies, that those who make a practice of operating by the Montgomery or Mayo method must be lucky if they do not meet with this accident occasionally.

Another case in which I operated by the Montgomery method gave me a good deal of anxiety and worry. A young married lady consulted me some years ago for constant backache, which rendered her a semi-invalid. An examination showed a markedly retro-flexed and enlarged uterus with a prolapsed and cystic right ovary. She had consulted several medical men in Australia and in London, and had undergone the usual treatment by curettage and pessaries without relief. Apparently the treatment by operation had never been seriously considered. After infinite trouble I persuaded her that an operation for replacing the uterus was the only means of permanent relief from her trouble. My opinion was confirmed by my friend the President of this Section. Eventually I fixed the uterus in position by the Montgomery method. She made a good recovery and her symptoms were entirely relieved. Five months later she returned from the country complaining of a return of all her old symptoms. As a matter of form I examined her and was astonished and horrified to find the uterus back in its old position. There was nothing for it but to admit that the operation had been a failure. She was naturally very much shocked and distressed, more especially as perhaps I had been over confident in prophesying that this operation could not fail to hold the uterus in position. She pluckily consented to let me make another attempt, this time I operated by Baldy's method, which has permanently kept the uterus in a forward position. She is now an active woman, able to enjoy horseback exercise and golf with impunity.

Intra-Abdominal Plication of the Round Ligaments by the Mann, Wylie, Dudley Method.

I have operated by Wylie's method in nine cases with two failures. It appears to me this method of operating for retro-displacements is only adequate in cases where the flexion is of recent date and not complete, but inadequate when the uterus has been completely retroflexed for some time, with attenuated and stretched ligaments and a heavy congested body.

In conclusion, my experiences of operation for retro-displacements of the uterus are as follow:—

Ventro-suspension.—116 cases, with 18 failures or faulty results.

Alexander's operation.—50 cases, with 2 failures.

Gilliam's operation and its modifications.—21 cases, with 1 failure and 1 accident.

Intra-abdominal plication of round ligaments.—9 cases, with 2 failures.

Baldy's operation.—127 cases, with 1 recorded failure through faulty technique.

The ventro-suspension operations were nearly all done before 1906.

The other operations have been only tabulated for the last five years.

I need hardly add that no operation for retro-displacement is complete without a thorough curettage of the uterus, and when necessary a repair of pelvic floor and repair of cervix.

DISCUSSION ON DR. McARTHUR'S and DR. HAMILTON'S PAPERS.

Dr. Thomas Murphy gave still another method of shortening the round ligaments in addition to the many methods already discussed, viz., folding the ligaments on themselves and stitching them to one another and to the upper surface of the uterus. This operation shared with Baldy's one good point, that the parietal peritoneum was not interfered with, and consequently there was less liability to future pain as a result. The greater sensitiveness of the parietal peritoneum was noticed here as elsewhere. In uncomplicated cases, without prolapse and without adhesions, he constantly performed external shortening with satisfactory results.

Dr. Chenhall felt a prejudice against Alexander's operation on account of ultimate bad results. Personally, he, on account of this prejudice, had rarely performed it, and, in the past, favoured Kelly's ventro-suspension method. He had not witnessed any of the bad effects attributed to the latter. Alexander's operation was sound in principle, and because he believed this he had adopted Gilliam's method in a large number of cases. In order to overcome the great objection against Gilliam's operation, that the round ligament was brought forward and away from its natural position, he adopted the plan of perforating the rectus muscle as far out as possible from the middle line. Then he made the smallest necessary opening in the wall. Thus he believed the risks of post-operative hernia were reduced. He always sutured the round ligament to the sheath of the rectus of the opposite side. In carefully selected cases he regarded the results as very satisfactory. He believed ventro-suspension a useful operation in correcting faulty retroversion of the uterus after removal of diseased Fallopian tubes, since it relieved pressure from the bowel and lifted the ovaries, when saved, into a better position. After all the eulogiums showered upon Alexander's operation to-day by experienced gynæcologists, his prejudice was not altogether warranted, and he would, in suitable cases, give it further trial.

Dr. BARRINGTON considered the ideal operation should have no mortality, should do the least anatomical damage, should stand the test of several pregnancies, and the uterus should be found maintaining its normal position years after it was performed. He thought that external shortening of the round ligaments in the inguinal canals in properly selected cases best fulfilled these desiderata. Before carrying out external shortening it was imperative to ascertain under the anæsthetic that there were no pelvic adhesions the result of infective salpingitis, and he never undertook the treatment of these cases without full permission to do what was found necessary. In performing an external shortening he always divided the inter-columnar fibres to enable the thorough stripping back of the peritoneal reflexion and the more efficient fixation of the ligaments. He had seen a fair number of women who had borne from one to four full-term children after this operation, and the uterus still maintained an excellent position in the pelvis. He emphatically condemned any suspensory operation when fertility remained. When there was doubt as to the presence of pelvic adhesions, or the appendix had to be removed, he preferred, by a transverse incision, to open the abdomen in the middle line for the intra-abdominal work, and then to shorten the round ligaments in the canals through the outer ends of the transverse incision. He usually shortened the round ligaments internally by folding them on themselves concertina-fashion, but the final position of the uterus was not so good as in external shortening, though the test of pregnancy had been successful. In external shortening the pubic end of the ligament was often found thin and wisp-like, but assumed decent proportions on running. The tendency of the weak pubic end to stretch probably accounted for the failure of some internal shortenings. Repair of the damaged pelvic floor was essential for permanency of results.

Dr. T. G. WILSON remarked that the subject of these papers was of considerable interest, and he wished to refer to a paper on this subject that he had presented at the Adelaide Congress in 1905. The cases recorded in that paper had tended to show that it was impossible to foretell what would happen to the uterus after the performance of a ventro-suspension or a ventro-fixation, especially if done with an absorbable material such as catgut. In the series recorded in that paper there had been eighteen recurrences of the retro-displacement out of thirty-five cases which were examined at varying periods of from six months to five years after the original operation. He had not done either of these operations in a child-bearing woman for the last six years, but had utilised the round ligaments to keep the uterus in position. In uncomplicated cases, he had had good results from the Alexander operation, and if one could be sure of the condition of the adnexa, he believed this to be the operation of choice. He had tried most of the other methods of shortening the round ligaments from within, and preferred the Baldy operation or a modified Gilliam; but, whenever possible, he used the transverse incision of Pflannelstein, and after completing whatever was necessary within the abdomen, he shortened the round ligaments from the ends of the incision after the method that had been referred to by some of the speakers as Mayo's method, but which he had been accustomed to speak of as Noble of Atalanta's method.

Drs. F. W. W. MORTON (Melbourne), PARKES (New Zealand), TAYLOR YOUNG (Sydney), and BURKETT (Dubbo), also joined in the discussion.

DISTRICT MIDWIFERY PRACTICE.

By E. LUDOWICI, M.B., C.M. (Syd.).

Hon. Assistant Surgeon, The Women's Hospital, Sydney.

THE object of this paper is to describe the principles on which the district work of the Women's Hospital, Sydney, is conducted, and to give some account of the work done in this department during the last seven years.

The hospital was founded in October, 1893, the founders being Dr. (now Sir) James Graham, Drs. A. Watson Munro and L. E. F. Neill, and Mr. David Fell, with Miss M. MacLeod as Matron. Dr. S. W. MacCulloch soon afterwards joined the staff; at that time systematic attendance on lying-in women in their own homes was almost entirely neglected, and one of the objects for which the hospital was founded is "to provide medical and nursing aid to poor women at their homes during accouchement."

The instructions to intending patients are as follows:—District patients must strictly obey the nursing instructions, and not allow any outside interference in the treatment: they are not booked before the seventh month, and those who apply before this time, except in the case of old patients, are informed that there is no obligation to attend them should anything occur before the seventh month.

Due inquiry is made as to previous difficulties or abnormalities (if any) in labour, albuminuria, &c., and they must be seen by the medical officer, and if necessary, undergo any treatment ordered; they must provide plenty of old linen, &c., washed and boiled, pay due attention to personal cleanliness; have ready a supply of bed-garments, &c., which have been boiled (this latter point is emphasised; as patients usually consider that new unboiled clothing is the proper thing). Water must be put on to boil when the messenger is sent for the nurse; this is usually at the first pain, or at the appearance of hæmorrhage or rupture of the membranes.

No telephone messages are taken, but a messenger must appear in person to take the nurse to the house; if the nurses have been called once and it proves a false alarm, they will go in the case of a second call in response to a telephone message, because they are prepared and are satisfied it is not a hoax.

The rules governing the nurses are as follows:—

The nurses reside at the hospital; they must not allow anyone to be present at their case or to help at their work other than those authorised to do so; the nurse to whom the case is allotted is held responsible for the proper nursing of the patient; each pupil nurse must furnish herself with a bag containing syringe and douche can, catheter, scissors, pins, needles and cotton, thread, ligatures, nailbrush, thermometer, inch tape, sterilised vaseline, corrosive sublimate tablets (tinted), cyllin, lysol, Jeyes' fluid, starch and boracic powder, saturated solution of sodium chloride, antiseptic soap, pair small scales, and matches; they must attend their patients for at least ten days after delivery, visiting them every day before noon, make entries in the day book and fill in chart notes immediately they return from their cases; they must report to the Matron immediately, by telephone or otherwise, the state of cases upon arrival; also, as soon as delivery is over, the nature of the case, and especially any abnormal symptoms occurring during the course of their attendance.

Two nurses are sent to each case; the senior has over six months' experience, and the assistant as a rule has received instruction in the wards. The senior nurse takes the fundus, &c., and the junior the baby; later on the senior allows the junior to take her place in order to gain the necessary experience. The nurses must remain with the patient for at least one hour after termination of the third stage, and see that all soiled things are removed from the room; it is sometimes found necessary to provide food from the hospital, also babies' clothing, and other necessaries. For an average of fourteen to twenty treatments daily it is found that a staff of from ten to twelve nurses is required, considerable time being occupied in going from house to house, and it is a question whether the provision of a vehicle would not be of great advantage by allowing of the treatment of a larger number of patients by the existing staff. The nurses' travelling expenses are paid by the hospital. The sister in charge of the district supervises, visiting the patient during the labour. During the puerperium she frequently visits the patient, examining patient, baby, clothes, and checking temperature, &c.

In the event of complications, such as prolonged labour, malpresentations, hæmorrhage, ruptured perineum, &c., the Resident Medical Officer is sent for, whilst the assistant honorary staff exercises a general supervision and undertakes any necessary operation. In cases where special treatment, as for albuminuria, sepsis, and other complications, is required, the patient is removed to the hospital.

Subsequently, in cases not requiring the presence of nurses, the patient is instructed to attend the out-patient department.

This short account cannot give anyone an adequate idea of the large amount of work performed in a quiet and unostentatious way.

Particulars of work done are:—

			Fothergill's Figures are—
Cases discharged	3,356		
Total deliveries	3,281		
,, vertex presentation	3,156	= 96·25 per cent.	of total deliveries.
,, ,, diagnosed	2,836		
L.O.A. position	1,961	= 69·14 ,,	of vertex diagnosed 72 per cent.
R.O.A. ,,	741	= 26·128 ,,	5 per cent.
R.O.P. ,,	100	= 3·526 ,,	21 ,,
L.O.P. ,,	34	= 1·19 ,,	2 ,,
Vertex position not defined	320		
Face presentation	8	= ·24 ,,	of total deliveries ·5 per cent.
R.M.P. position	1		
L.M.P. ,,	3		
L.M.A. ,,	2		
R.M.A. ,,	2		
Pelvic presentation	109	= 3·322 per cent.	of total deliveries 3 per cent.
L.S.A. position	52		
R.S.A. ,,	18		
R.S.P. ,,	7		
L.S.P. ,,	11		
Positions not defined	22		
Transverse presentations	6	= ·18 per cent.	4 per cent.
L.A.A.	1		
R.A.	2		
Undefined	3		

(You will notice that these sets of figures do not agree, as my statistics show that R.O.A. accounts for 26 per cent., and R.O.P. 3½ per cent., whereas text-book figures show for these 5 per cent. and 21 per cent., respectively.)

Funis presentation	2
Induced labour	5
Forceps used	99
Ruptured perineum	227
,, Cervix	1
,, uterus	2
Adherent membrane and retained placenta	101
Placenta prævia	6
Accidental hæmorrhage	8
Ante-partum ,,	29

Hæmorrhage, third stage ...	48	
Post-partum hæmorrhage ...	105	
Secondary hæmorrhage ...	7	
Twins	31	
Infants died	28	
„ deadborn	26	
„ premature	77	
„ still-born (including premature)	75	
Abortions	86	
Albuminuria	78	
Eclampsia	12	
Phlegmasia	7	
Mastitis and fissured nipples	50	
Septicæmia following premature labour	1	Before arrival of nurses.
Sapræmia	125	
Pneumonia	1	
Vulvar abscess	1	
Puerperal mania	7	
Prolapse cord	2	
Spina bifida	1	
Bronchitis	1	
Hydrocephalus	4	
Anencephalic fœtus	2	
Hydramnios	8	
Pulmonary embolism ...	3	
Imperforate anus	1	
Deformed pelvis	2	
Double talipes varus ...	1	
Ophthalmia	8	
Hydatidiform mole	2	
Infant died from hæmorrhage of bowel and umbilicus ...	1	
Hæmorrhage from bowel (infant)—recovered ...	1	
Subcutaneous emphysema ...	1	

Deaths, 5 in 3,281, = 1 in 656·2—

1 Eclampsia.
1 Ruptured uterus.
1 Eclampsia before and after labour.
1 Hæmorrhage and sapræmia.
1 Pulmonary embolism.

If we include 1 septicæmia in case of premature labour where baby was born before arrival of nurses, the proportion is 1 in 546·8.

The indoor training of midwifery nurses is greatly supplemented by the district, for the trainees learn to conduct cases without the many accessories which an hospital provides, and which must be dispensed with in private homes; they also are educated to assume the responsibility that a nurse must take in work where a doctor is not always available for the various and sometimes serious emergencies.

Students learn under practical conditions what they must expect in the exercise of their profession, although undoubtedly the time allotted by the curriculum is too short for one of the most important branches of medicine, and thus affords foundation to a well-known statement that bad midwifery is responsible for the large amount of gynæcological surgery done nowadays.

Poor women are taught the rudiments of asepsis in the most practical way possible, and the importance both of proper treatment of themselves during the most important period in a woman's life, and of the care of the infant, especially in regard to breast feeding; the value of these instructions is shown by the fact that when the nurses are called to attend a woman on a second occasion, there is considerably less difficulty in preparing both the patient and the room; it is remarkable to see the transformation effected by the nurses—a squalid, filthy room being transformed into a neat and tidy one, where any operation could be performed.

It must be plain to everybody that treatment at home as set out above is not prejudicial to the patient, and, while being consonant with the natural maternal desire to keep in touch with the various household affairs, care of children, &c., makes way for those cases which must be taken from home, and relieves to some extent the State and charitably disposed of the expense entailed by the maintenance of hospitals.

DISCUSSION ON DR. LUDOWICI'S PAPER.

Dr. Meyer regarded Dr. Ludowici's paper of real practical value. In connection with the Women's Hospital, Melbourne, the President (Dr. Adam) and himself had for a long time advocated an extern-midwifery department, and in the four and a half years of its progress it had proved very satisfactory. The advantages of the system were obvious. It was a boon to women who had the comfort of being in their own homes; it was a gain in State economics; it did not pauperise to the extent of hospital treatment; and it made for a fuller and more practical education of students in obstetrics, as a complement to their hospital experience. Lying-in hospitals were a necessity, but he was opposed to the principle of multiplying them in the face of the benefits of a well-organised extern-midwifery system.

SOME RESULTS OF LACERATION OF THE CERVIX.

By Thomas Murphy, M.A., M.D., M.S. (Melb.), F.R.C.S., I.

In this paper I propose to deal with some less commonly-discussed results of unhealed lacerations of the cervix uteri—that is, of lacerations in which either the torn surfaces have not come into apposition and become united, or in which the exposed and torn surfaces have not healed by granulation and become completely covered with stratified squamous epithelium. If the laceration has healed by either of the above methods, and if there is no marked ectropion, so far from causing any ill-effects, it may be of some advantage to the patient by facilitating drainage of the uterine mucous membrane. If the torn surfaces have failed to heal wholly or in part within a reasonable time, the unhealed surfaces, together with the exposed and everted cervical mucous membrane, present the usual raw-red granular discharging surface, the so-called erosion. This eroded or catarrhal surface is, in some cases, covered with columnar epithelium, and numerous newly-formed glands open on the eroded surface. In some cases this columnar epithelium is wanting in parts, and then is mainly discernible where the glands open on the surface. In some cases part of the eroded surface is formed by granulation tissue only. These red catarrhal patches are inlets of infection, and this infection, slow and chronic though it is in the cases under review, is the cause of most of the morbidity that results therefrom.

Passing over the effects which these unhealed lacerations may produce on the cervix, the uterine body, the tubes, or the adjacent peritoneum, I shall deal with some more remote, but common, results.

After the infection has spread through the cervix, it may cause the same changes in the parametrium or pelvic cellular tissue which it causes in the cervix. Just as in these neglected cases we often get a condition of chronic cervicitis, so also we often get a condition of chronic parametritis.

The condition I refer to has nothing to do with acute cellulitis, and is essentially chronic from the beginning. Not much is written about it in ordinary text-books. Some do not describe it at all. Some describe it as resulting only from acute cellulitis, and as causing no symptoms. One of the objects of this paper is to show that it is a common disease, and that it often causes marked symptoms. The infection, when it has reached the parametrium, spreads along the lymphatics, causing chronic lymphangitis, with some infiltration, thickening, swelling, and tenderness along the course of these vessels. This in time goes on to sclerosis of the affected parts, with consequent hardening, stiffness, and want of elasticity. The elastic and muscular elements in the cellular tissue are more or less atrophied and replaced by tougher fibrous tissue. This condition of infiltration, resulting in sclerosis, is liable to exacerbations from time to time, whenever the eroded surface is the seat of a fresh infection. Its mildness and chronicity are probably due to the excellent drainage afforded by the dependent position of the cervix. The sclerosis of the cellular tissue may be sufficient to compress the lymphatics, veins, or nerves which pass through it, or to cause interference with the functions of the viscera which are embedded in it. This may cause damming back of the lymph or blood-stream, with thickening and enlargement of the cervix. When the infundibulo pelvic ligament is affected, it may cause varix in the broad ligament and changes in the ovary. The sclerosed tissue, through want of elasticity, may interfere with the action of the bladder, ureter, or rectum.

Chronic parametritis may, in marked cases, be easily felt by the palpating finger. In less marked cases it can be felt by expert palpation only; and in another class of cases it cannot be felt at all, but its presence can be diagnosed by pain caused when the affected tissue is stretched by the examining fingers. One of the commonest positions in which it is found is at the side of the cervix, the hardened tissue spreading outwards towards the pelvic wall along the line of the uterine vessels. Another common position in which it can be felt by rectal or vaginal palpation is in the utero-sacral ligaments. Other parts of the parametrium are not so accessible to touch, but the sclerosis can often be demonstrated during abdominal operations. It is a common cause of pain on one or other side of the lower abdomen during exertion in the erect position, or on jerking, jarring movements. This pain is caused by the descent of the uterus during such movements. It is not due to the weight of the uterus only, as is commonly asserted, but is due to intra-abdominal pressure during effort forcing the uterus downwards and dragging on the stiffened ligament. Backache is a common symptom when the utero-sacral ligament is affected on one or both sides, and in this position there may be pain on coitus, and discomfort on defæcation. When the tissue between the uterus and the pubes is sclerosed, such as is sometimes seen, for instance, in retro-displacements of the uterus, distension of the bladder may cause painful stretching of the sclerosed tissue, and irritability of this organ may be a marked symptom.

These symptoms may vary from time to time, according to whether the inflamed ligaments are allowed to become quiescent, or whether fresh supplies of infection are admitted to cause further irritation.

The tenderness usually felt on stretching the affected tissues may lead to errors in diagnosis; for instance, if the posterior radiations of the parametrium are sclerosed, the examining finger, on lifting the cervix forward, may cause severe pain. This pain may easily be attributed to a tender cervix or uterine body. If the infundibulo pelvic ligament is stretched on examining the ovary bi-manually, chronic parametritis in this part may lead to a diagnosis of a diseased or tender ovary. Diminution of elasticity in the region of the bladder may lead to a diagnosis of cystitis, or to the impression that the uterus is the sole offending organ by either pressing upon or dragging upon the bladder. Where pain is complained of on exertion, or on putting the ligament on the stretch by the examining finger, if, in addition, no diseased condition can be palpated in any of the organs, the patient may be classed as a neurotic. The pain felt by putting tension on the affected ligaments is somewhat analogous to the pain felt on moving the stiffened ligaments of a rheumatic joint.

In extreme cases there is more or less fixity of the uterus, and in some of these this organ is drawn slightly to the affected side. When the sclerosis affects the broad ligament on either side of the cervix, it may cause some narrowing or distortion or kinking of the ureter. This condition is usually accentuated by some of the concomitant results of the torn cervix, such as displacements. Here we may have some interference with the drainage of the ureter, with the result that the kidney is compelled to secrete urine under higher pressure than usual. This may lead in time to chronic granular kidney, with high blood-pressure and a whole train of attendant symptoms.

The gulf between an abnormal uterus and an apoplectic stroke is a wide one, but I have seen it bridged over in a case of myoma of the uterus of moderate size in a comparatively young patient. The subjective symptoms attributed to chronic parametritis may be caused by many other morbid conditions. The object of this paper is to show that in more cases than are usually recognised such symptoms are due to chronic inflammation of the pelvic cellular tissue, having its origin in absorption from a chronically-infected lacerated cervix.

A chronic catarrhal or quasi-ulcerated patch of similar size would not long be tolerated at the urinary meatus, at the angle of the mouth, or at the meatus of the nose; and, were it not for the fact that the cervix is almost devoid of ordinary sensation and completely hidden from view, these chronic and often septic lacerations would not be so often neglected.

Another more remote result of septic absorption from the torn cervix in these cases is a chronic, mild toxæmia, variable from time to time with the amount of infection admitted by the cervical lesion. Headache, general malaise, and a certain amount of anæmia, in addition to the ordinary pelvic symptoms, may result. Sometimes so-called bilious attacks are due to an intermittent dose of poison from this source.

Many cases of rheumatoid arthritis, and of other arthritic affections, are due to chronic absorption from septic foci in various parts of the body. Such parts as the alveoli of the teeth, the lachrymal sac, the middle ear, and the accessory sinuses of the nose are carefully examined to find the point of entrance of infection. These rheumatoid manifestations in joints and other parts are much more common in women than in men. One authority gives the ratio as 411 to 89 in a long series of cases, and in older books multiple childbearing was given as a cause of the disease. These arthritides are found in the class of women in which unhealed lacerations are common. By bringing about healing of these raw surfaces, which usually consist of a partially-healed laceration and an everted cervical mucous membrane, some of these blood and joint conditions may be prevented. Even after the joint condition is established the disease may be arrested. In old cases of chronic parametritis much can be done. Some sclerosis will remain, but the tissues become softer, more elastic, less dense, and less painful when the avenues of infection are permanently closed.

To sum up, the points I have attempted to bring out in this paper are:—

(1) Chronic parametritis, as a result of old lacerations of the cervix, is common, but often not recognised as such.
(2) Variation of symptoms from time to time, due to variable supply of infection.
(3) Renal complications may result from this chronic parametritis.
(4) Certain blood and joint diseases, the result of toxæmia, may be relieved by treating these old cervical lacerations.

Drs. Chenhall (Sydney), Parkes (N.Z.), and J. A. G. Hamilton (Adelaide) discussed the paper.

THREE CASES OF MAL-DEVELOPMENT OF THE UTERUS.

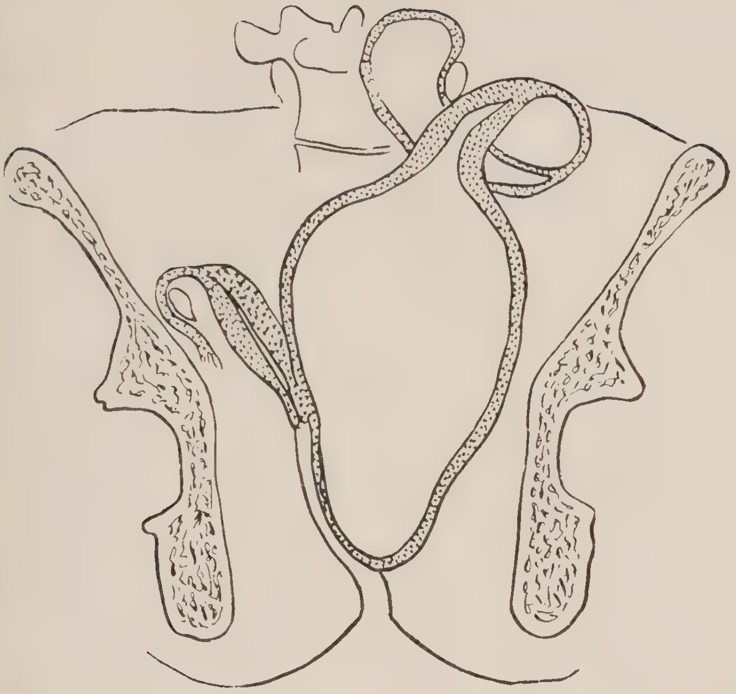
By T. G. Wilson, M.D., C.M. (Syd.), F.R.C.S. (Ed.).
Hon. Gynæcologist, Adelaide Hospital, S.A.

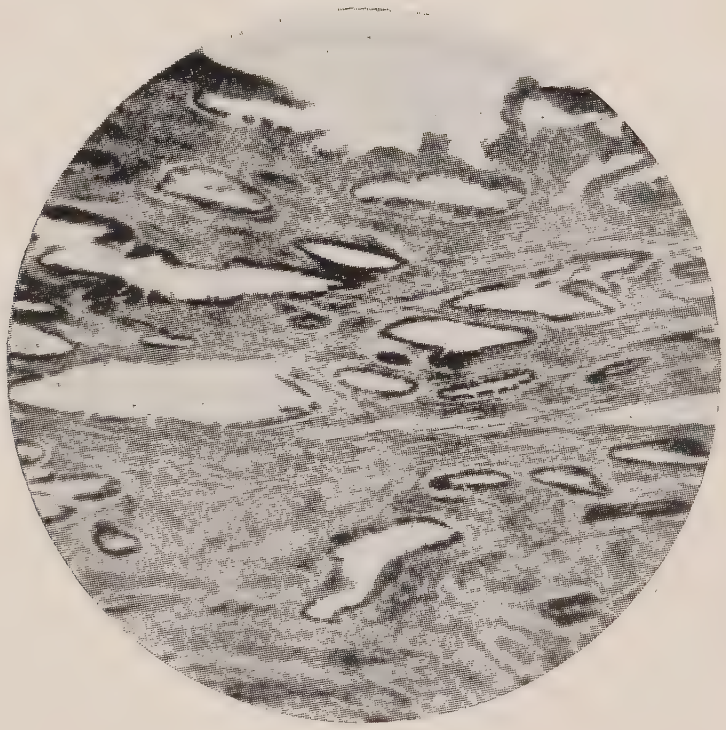
The female genital organs, like other parts of the body, being liable to errors of development, marked or slight, it behoves us to be on the watch for, and able to recognise, such signs and symptoms as are connected with these conditions. While the slighter degrees of mal-development are not uncommon, the more marked types—such as double uterus—are comparatively rare; but there are certain conditions, dependent on these errors of development, which make it necessary to bear the possibility of their occurrence in mind, and in some cases the symptoms they give rise to are so marked that they might almost be called pathognomonic. Take, for instance, superfœtation—a condition now generally held to be only possible when there is a double uterus—and also some of the cases where menstruation continues throughout pregnancy, which are due to the same condition. Again, the various cystic swellings arising from atresia of some part of the genital canal, or dilatation of fœtal structures which should have become obsolescent, sometimes behave in such a definite manner that their recognition should not be difficult if we bear in mind the morphological evolution of the genital tract, as the knowledge of this evolution will, in the greater number of cases, enable us to give a satisfactory explanation of the mode of origin of these various conditions. So there is a definite interest in these cases, both from the clinical and the scientific point of view, and it is for this reason that I venture to record details of the three following cases which it has been my good fortune to have under my care.

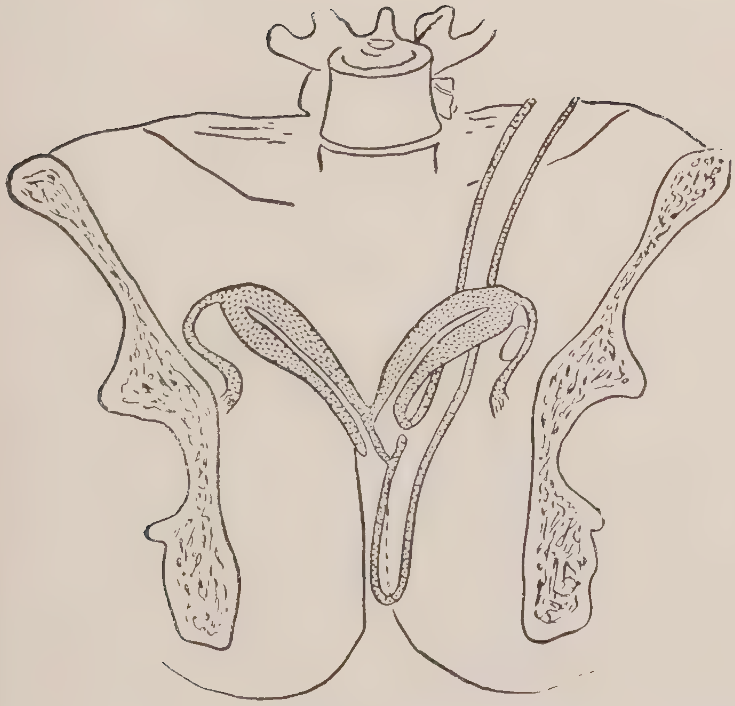
Case 1.—A.B., a girl aged 14, a patient of Dr. Hone, of the Semaphore, with whom I saw the patient in consultation on 29th November, 1907, and whose history was as follows:—She complained of increasing pain at the time of her periods, and a lump in the abdomen. Her menstrual life started at 13½ years; the flow had come regularly, but with increasing amount of pain each time, and with the last period there had been slight pyrexia, and at this time Dr. Hone had discovered a central abdominal swelling. At 10 years of age this patient had had what was looked on and treated as a gonorrhœal vaginitis. Examination of the abdomen disclosed a cystic swelling extending almost to the umbilicus; to the right of this was a hard mass the size of a mandarin, while to the left was another softer mass as large as an orange, and both of these masses apparently connected with the central tumour. Vaginal examination, under ether, disclosed a large cystic swelling continuous with the mass felt above, bulging out the left vaginal wall and reaching almost to the introitus, so that the vagina was reduced to a narrow, antero-posterior slit, at the extreme upper end of which could be felt a small conical cervix.

I opened the abdomen in the mid-line, and found the mass felt on the right side was a small uterus, with normal ovary and tube attached. The large central mass, which bulged below almost to the vulva, was the size of an adult skull; on top of it, and to the left, was perched another uterine body, the tube of which, at its outer end, was distended to the size of a large orange, with the ovary attached below. There had been some leakage of blood from the fimbriated end of the tube, which was adherent to the sigmoid. The central swelling was opened from the vagina, and two and a half pints of tarry blood evacuated. Then from abdomen a left hysterectomy was done; but, as the walls of the cyst projected to a low level laterally, and down into the paravaginal tissues on the left side, it was not practicable to remove the whole of this part of the cyst, so the edges of the cyst-wall left were stitched together and the raw surface left covered with bladder peritoneum. A drainage tube was inserted into the cavity left through the incision made in the vagina, and the abdominal wound closed. The patient's recovery was satisfactory, but the sinus left in the vagina continued to discharge thick, mucoid material, eventually becoming purulent. This sinus has persisted up to the present time, despite all sorts of treatment, including the use of bismuth paste, which Dr. Hone tells me seemed to lessen the discharge for a time. Dr. Hone kindly sent this patient to see me again recently, and now, four years after operation, there is still a sinus, 1 inch in length, at the vault of the vagina to the left of the cervix, which is discharging purulent fluid. The patient's health is good, and menstruation normal; but in view of the possibilities of infection, should pregnancy occur at any time, I have suggested that the remaining portion of the cervix should be removed.

I have made a sketch of the condition as found at operation, and it will be seen that the condition suggests a double uterus, with a hæmatocolpos of the left side. Dr. De Crespigny has kindly cut sections of the wall of the lower portions of this cyst, and Dr. Mouldon has taken a micro-photograph of one of these, from which it will be seen that the lining of this portion consists of cervical endometrium, the glands of which, instead of running at right-angles to the surface, are parallel to it, apparently the result of distension pressure. So that the large cyst found in this case was really cervical in origin, forming a hæmatotrachelos, which had been distended sufficiently to hold 2½ pints. The distension that occurred







here, of what corresponds to the lower uterine segment, is probably to be explained as being analogous to the action of this passive segment of the uterus during labour, as the upper segment, the fundus proper, was perched on top of the distended cervix, and practically hardly distended at all. That this amount of distension of the cervical portion does take place is of interest, as in such a case it would be easy to mistake this for a distended second vagina. The walls of this distended portion merged gradually into the uterine cavity proper, and the character of the discharge which has persisted so long is what one would expect from a glandular surface, and not from vaginal mucous membrane, which, as we know, is practically devoid of glands. The history of a gonorrhœal infection in this case suggests the possibility of the atresia here as being acquired, and not congenital, as such cases generally are; but there was no sign whatever of a cervical orifice, and I am inclined to regard it as a congenital condition.

The second and third cases were under my care recently, being seen first on two successive days. The second case was diagnosed after examination; the third not till the abdomen was opened.

Case 2.—M.F., æt. 14 years, was seen in January, 1911, with Dr. C. Magarey, of Norwood, and she complained of having a profuse dark blood-stained discharge, which had come on suddenly five days previously. There had been no pain, but she has had intermittent fever since the flow started. Menstruation had started at 13½, coming regularly, and with only slight pain. Vaginal examination, under ether, disclosed a small cervix, to be felt high up on the right side, and a small uterus continuous with it. To the left of the cervix was a small opening, from which there was a discharge of foul-smelling blood-stained fluid; and another body, feeling like a second uterus, was felt on the left side. This opening was enlarged, and a drainage tube inserted. After this the patient kept up an intermittent fever for three weeks, and though drainage was free the discharge did not lessen, and still kept offensive. I therefore opened the abdomen, and the condition found was as follows:—A small uterus, with normal tube and ovary on the right side; a somewhat bigger uterine body, with normal tube and ovary, on the left side; and attached at the level of the isthmus of the left uterus was a longitudinal swelling slightly thicker than one's thumb, running upwards in the position of the left ureter, and retro-peritoneal in position. The left ureter was located to the left of and behind this swelling. I removed the left uterus, and in doing so found that the lumen of this longitudinal swelling communicated with the cervical canal of the left uterus by an opening the size of a lead-pencil, and on pressing the longitudinal swelling from above dark, tarry, offensive fluid could be squeezed out. A probe could be passed up in the lumen of this tube for a distance of 5 inches. The lumen of this tube admitted the little finger, and on passing the finger downwards it was found that there was a continuation of this lumen downwards between the bladder in front and the anterior vaginal wall behind to about half an inch above the urethral orifice, and this cavity was also partly filled with the same dark, tarry fluid. I stitched the edges of this upper tube to the vaginal vault, and opened the lower end of the continuation downwards through the anterior vaginal wall and inserted a rubber tube, first attempting to destroy the lining membrane of this succulus. The peritoneum was brought over the raw surface left by the removal of the uterus, and the abdomen closed. I did not think it wise to attempt the removal of the longitudinal swelling, owing to the infected nature of the discharge, as it would have necessitated opening up fresh areas of cellular tissue. This patient did well, the discharge from the vagina and from the tube ceasing after about ten days, and she is now well and menstruates normally.

I append a diagram of the condition as found at operation. The mode of origin of this elongated tube communicating with the cervix on the left side is interesting, and, from a developmental point of view, suggests two modes of origin. First, it might be explained as being developed from a double Mullerian duct, or as a diverticulum from the duct of the left side. This is the usual explanation of these so-called accessory uteri, and if this be the case it would approximate somewhat with a case recorded by Depage, and mentioned in Allbutt and Playfair's "System of Gynæcology," where the condition is described as "Trifid Uterus," and the authors remark that "these anomalies fully deserve to be called malformation 'by excess,' which the so-called 'double' uterus does not." Secondly, the condition might be explained as being a persistent Wolffian duct, which came to communicate with the Mullerian duct of the left side, probably at the point where these two ducts cross each other, as in early embryonic life the Mullerian ducts are originally external to the Wolffian ducts, but later grows inwards preparatory to the process of fusion to form the uterus. Granted the persistence of this duct, and also a communication with the left Mullerian duct, when the left uterus began to distend the menstrual blood would be forced into this tube also. Another point in favour of this possible mode of origin is the fact that the lower end of this tube extended down in the anterior vaginal wall, in the position of Skenes' tubules, which we know are normally developed from the lower end of the Wolffian ducts in the female, and occasionally in the human, as normally in the sow and some other vertebrates; the parovarian tubules, which are also developed from the remains of the lower end of the Wolffian duct, can be traced to this position in the vagina. I regret that I did not remove a portion of this tube for microscopic examination.

Case 3.—H.R., æt. 22, was sent into the Adelaide Hospital by Dr. Plummer. She complained of an abdominal tumour, and of having had increasing pain at the time of her periods for the last six months. Menstruation started at 16 years of age, was regular, and, till six months previously, had been painless. In the abdomen was a cystic swelling, median in position, and extending almost to the umbilicus. Pelvic examination showed that this tumour bulged into the vagina, especially on the left side, and the cervix could be felt high up on the right side, but the uterine body could not be differentiated from the tumour mass. On opening the abdomen the condition was at once evident. There was a small uterine body, with normal tube and ovary pushed over to the right side of the pelvis, and perched on top and to the right of the central tumour mass was another uterine body, with normal ovary and tube attached. In this case, as in No.

1, the central cystic mass was apparently the dilated cervix. From the vagina an opening high up close to the cervix was made, and nearly 2 pints of dark, tarry fluid evacuated. With a sound passed into the right uterine cavity it could be appreciated that there was merely a thin partition between the right cervical canal and the left cyst cavity. This partition was cut through at its lower portion, and the vaginal incision prolonged to open into the cervical canal, and the mucous membrane of the dilated cervix stitched to vaginal mucous membrane anteriorly and posteriorly, and a self-retaining rubber catheter introduced through this incision. On examining the condition again from the abdomen, the left uterus had now come down into the pelvis, and was alongside the right uterus. The abdominal incision was closed, and the patient made a satisfactory recovery, the tube being removed at the end of a week, during which time the cavity was washed out frequently. The vaginal sutures were removed in three weeks, the common cervix now being a transverse slit three-quarters of an inch long, the sound passing 2½ inches into the right uterus, and 3 inches into the left. This patient has since menstruated normally, and is quite well at present.

This case was an example of Uterus Bicornis, with atresia of the left side and resulting hæmato-trachelos. It is rather remarkable in this case, if the atresia were congenital, that she should have menstruated normally from 16 till 21½ years of age without any symptoms of obstruction. Probably the treatment adopted in this case might have sufficed in case No. 1, but in that case the presence of the distended tube, with leakage of blood and inflammatory adhesions, decided me in removing the uterus and adnexa of that side.

SOME NOTES ON EARLY RISING DURING THE PUERPERIUM.

By R. Tate Sutherland, M.D. (Melb.), C.M. (Glas.).
Honorary Obstetric Surgeon, Women's Hospital, Melbourne.

Early rising in the normal puerperium is no new idea. Quite apart from the classical case of the savage woman who has her baby without lying up at all, it has been advocated at varying intervals in various countries during the last seventy years, but until recently it has not been considered seriously by the general body of the profession. Haultain's observation, however, brought the subject so prominently before the world that one had to seriously consider the position to ascertain if, in the progress of things medical and surgical, obstetricians had been blindly following tradition when they should have been working out new ideas, or whether the rules and regulations governing child-bed which had been handed down the centuries were not the best.

Rapid progression has been the rule all along the line, and especially is this the case in surgery. The after treatment of abdominal surgery is very different now to what it was, say, ten years ago, and the surgeon's thought now is not how long he must keep his patient in bed but rather how soon he can get his patient up. This idea has subconsciously thrust itself on obstetricians, and the thought that we should keep a woman in one room for four weeks after parturition does not now often occur to us. This period is, however, laid down in some of the latest and best text-books—two weeks in bed, one week on a lounge, one week walking about the room, and then she is allowed downstairs.

There are six complications which the obstetric surgeon has always in his mind during the puerperium, viz.:—

1. Post-partum hæmorrhage.
2. Puerperal infection.
3. Retro displacements.
4. Prolapse.
5. Phlegmasia alba dolens.
6. Embolism of the lung.

The question at issue is: Will prolonged rest in bed prevent these complications, and will early rising tend to their occurrence? These are the points on which one requires to be convinced before an opinion can be given. It is my firm belief that early rising amongst the working classes has been the rule for many years, but quite without the sanction or knowledge of attending obstetricians, as may be instanced by an interesting conversation I had a few weeks ago with an unqualified midwifery nurse, who, however, has a large experience, and is never unemployed. She said: "I always get my patients out of bed on to a chair on the third day while I air and make the bed, and I allow them to stay up till they feel tired. I find the lochial flow is increased, and very often clots are passed, and that patients treated in this way have a much more satisfactory puerpurium, and are better and stronger at the end of a fortnight than those who are kept in bed for ten days or longer. They feel stronger and healthier and are able to resume their ordinary household work without being fatigued. I based my conclusion on the fact that when visiting the houses of the poor I often found women sitting on the edge of the bed on the third day washing their towels, and these women, contrary to all teaching, did so well that I followed out the early-rising idea on my own patients." "But," I said, "when you are nursing a case for a doctor who wishes his patient kept in bed for ten days or a fortnight, what do you do?" to which she replied, "the doctor may think his patient remains in bed, but every case I nurse gets up on the third day if I consider she is well enough."

Haultain, of Edinburgh, is the latest to advocate early rising, or, as he terms it, "the rational puerperium," and he advises that all normal cases should sit out of bed on the second day for an hour; for an hour twice a day on the third day, and so on. He defines normal cases as those in which the labour has been normal in every way, without any mechanical interference, such as forceps or turning, where there is no perineal laceration, adherent placenta, or post-partum hæmorrhage. This method was adopted in 100 chosen cases at the Women's Hospital, and with apparently quite satisfactory results as far as the patients were concerned; but in a large hospital it was found to be very troublesome to pick out these special cases day by day, so a more easily-worked plan was adopted, by allowing all cases except those who had had much manipulative treatment, ante or severe post-partum hæmorrhage, or considerable perineal laceration, to sit up in bed to meals on the third day, and to rise and sit out in a chair on the fourth day for an hour, the time out of bed being gradually lengthened day by day. These cases included slight perineal tears which have been carefully sutured by silkworm-gut and cat-gut, and those cases which had required forceps. The general idea was to get as many patients as possible up on the fourth day, but if from any circumstances it was deemed inadvisable to do so they were kept in bed as long as was considered necessary.

There is no doubt that as far as the feelings of the patients are concerned, they prefer early rising, and when discharged on the tenth or eleventh day they are in a better physical condition to return to their homes and duties than those who are discharged from the hospital after an equal length of residence, but who have been kept in bed up to the day before leaving. Involution proceeds just as satisfactorily, and there is less likelihood of retention of clots and lochia.

For statistical purposes, I took 200 consecutive cases of early rising from the records of the Women's Hospital, the date of rising ranging from the fourth to the eighth day.

Of these 118 sat out on the fourth day.
38 ,, fifth day.
17 ,, sixth day.
10 ,, seventh day.
17 ,, eighth day.
200

2 were discharged on the ninth day.
116 ,, ,, tenth day.
37 ,, ,, eleventh day.
19 ,, ,, twelfth day.
14 ,, ,, thirteenth day.
3 ,, ,, fourteenth day.
8 ,, ,, fifteenth day.
1 was ,, ,, seventeenth day.
200

One case returned after ten days with uterine hæmorrhage; a very small piece of placenta was found at the fundus, and removed by the curette. She was quite well after.

One case fainted on sitting up in bed on seventh day.

One case was giddy on sitting out on fourth day.

One case complained of backache on sitting up in bed on second day.

One case died suddenly of pulmonary embolism on the thirteenth day whilst being discharged in the waiting-room. She sat up on the seventh day, but had a temperature on the evenings of ninth and tenth days of 103, and was then kept in bed.

One case complained of headache on the eighth day (sat out fourth day).

One case had shreds on tenth day (sat out sixth and discharged thirteenth day).

One case had placental tissue removed on the ninth day.

One case had triplets.

Three cases were twins.

Eight cases had forceps (4 per cent.).

Five cases had post-partum hæmorrhage at time of confinement (2·5 per cent.).

One case had ante-partum hæmorrhage.

Four cases had inflamed breasts (2 per cent.).

Twenty-eight cases required a uterine douche (14 per cent.).

Sixty-one cases (30·5 per cent.) had a temperature of 99 F. or over at some period or other during the puerperium; but if the cases are excluded in which the temperature was under 100 F., there remain thirty cases (15 per cent.) with temperatures of 100 F. or over at some period.

If we exclude forceps, torn perinei, and inflamed breasts, and take only cases of normal labour, the cases with temperatures of 99 F. or over are reduced to 40 (20 per cent.); while if we take the cases of normal labour, the temperatures of 100 or over are 15 (7·5 per cent.). This is including inflamed breasts.

In private practice, my custom with patients in a private hospital is to allow them to sit up in bed to meals on the third day, and then to sit up in bed whenever they feel inclined up to the sixth day, when they are allowed to sit out of bed in an easy chair for an hour, the period being prolonged from day to day, the patient being carefully watched for any untoward symptom.

In private houses I am more conservative, and keep them in bed for eight days, because without hospital discipline one never knows how much a privilege may be abused.

Retro-displacements of the uterus do not manifest themselves until some time after the patient has been up and about, and no cases of this were noted in the series up to the time of leaving the hospital. Whether any occurred subsequently I am unable to say, but my opinion is that early rising is not productive of it.

Conclusions.

We may, therefore, form the following conclusions:—

1. That the general health is in no way disturbed by early rising.
2. That patients, as a rule, prefer it to prolonged rest in bed.
3. That in hospital practice, the patients leave the institution better equipped to perform their household work.
4. That in private houses rising before the eighth day is undesirable.
5. That post-partum hæmorrhage is not induced by early rising, and involution is satisfactory.
6. That the risk of puerperal infection is not increased.
7. That, so far as we can say, the liability to retro-displacement and prolapse is not increased, and the same thing may be said of phelgmasia, alba, dolens, and embolism of the lung.

DISCUSSION ON EARLY RISING DURING THE PUERPERIUM.

Dr. **Felix Meyer** said that, however much in theory one might be opposed to a departure from the idea of keeping a woman in bed after delivery for a definite period, experience such as that recorded by Dr. Tate Sutherland must be accepted in favour of lessening the usual period of rest. But he was not satisfied that the real effect of this shortened rest could be estimated a week or even a fortnight after delivery. A heavy uterus dragging on subinvoluted ligaments tended to retro-position and prolapse. Long experience as a resident surgeon in a Women's Hospital had convinced him that women who got up and went about their work in a week or little more developed these and other pelvic disabilities; and there was authoritative statement that they were fairly common among certain native women (Maoris and Bosjesman) who practised very early rising in the puerperium. He would not attribute the fatal case mentioned by Dr. Sutherland to this course; he regarded the embolism as of septic origin. In Vienna last year, he had inquired as to the results of allowing women to go about a few days after delivery; but here again, though the general effect on their health was good, evidence was not forthcoming as to the resulting condition of the pelvic organs at a more remote period. In bringing forward this subject, Dr. Sutherland had furnished material both of great interest and importance.

Dr. **A. Norman McArthur** said: " There has been much written in American and Continental journals advocating very early rising in the puerperium, and it is pleasing to see that Dr. Tate Sutherland, in his very practical paper, has shown some moderation in that treatment. It is an effective method of drainage without doubt, but much can also be done by elevation of the head of the bed, and change of position of the patient. The suspensory ligaments of the uterus being retro-muscular involute with the uterus, and before involution is completed much weight is brought upon them, perhaps with dire results. Pulmonary embolism very rarely ever occurs, and is probably not influenced in the slightest by early rising. He did not think with Dr. Meyer, that pulmonary embolism always occurs in septic cases or anæmia. The only case he ever saw of pulmonary embolism, there was absolutely no sign or symptom of anæmia of Lepais before her death.

Dr. **Samuelson** (Bangalow) said that it was unnecessary to discuss cases abnormal, of which each must be taken on its merits; but of cases normal he believed that retro-deviation argued slackness, and as no woman gained " tone " in bed, he advocated rising early—viz., within one week. He believed that there was more retro-deviation in " hay-bag " obstetrics, with its nine days' fetish worship, than in the more scientific. He also frequently found cases with temperature showing no further abnormality in which the temperature would not fall until the patient rose; in other words, the uterus was normally placed and normally drained.

Dr. T. G. **Wilson** was glad this subject had been brought up, as he had been following much the same lines as were laid down in Dr. Tate Sutherland's paper for the last two years; but he thought that a good deal of misconception in regard to getting patients up early had arisen. Personally, he always made a point of telling the patient and her friends that, although she was allowed out of bed earlier, it did not mean that she would be allowed to go about and undertake her household duties any earlier than under the old régime. He thought that the cases should be chosen, and in the absence of any laceration or stitches, or any complication ante-partum, he allowed the patient to sit up to meals on the second or third day, and to sit on a chair for an hour or so each day after. He felt sure that the lochial drainage was better, and even before he had adopted the early-rising method had always had the head of the bed raised for this reason. Many patients, such as one meets in a maternity charity, prefer to stay in bed longer, and with some of these the lying-in period is the only chance of a rest they have each year. In this class of case, if the patient feels she would sooner rest, he would not ask her to rise early; but if the patient feels fit and strong enough, he can see no reason why she should not get out earlier, and in his own experience he had seen no ill results arising from the practice, but rather thought patients progressed better and involution was more satisfactory.

Drs. S. H. **MacCulloch** (Sydney), **Scott** (Hobart), and **Taylor Young** (Sydney) discussed the paper. The **President** summed up, and Dr. **Tate-Sutherland** replied.

Afterwards the President demonstrated a most interesting specimen of ovarian pregnancy from a case on which he had recently operated.

"BRYCE-TEACHER OVUM."

A lantern demonstration in the Anatomy Lecture-room on "The Bryce-Teacher Ovum," by Professor J. T. Wilson and Dr. H. C. Taylor Young opened the proceedings on Friday morning. Dr. Taylor Young gave the history of the specimen, and Professor Wilson demonstrated the slides. This demonstration was undoubtedly the event of the Section, and at its close the applause was loud and prolonged.

The Section then reassembled in the Medical Lecture-room, and before the remaining business on the programme was proceeded with it was decided to send the hearty thanks of the Section to Dr. Teacher, Glasgow, for sending the splendid lantern slides.

ECLAMPSIA.

R. H. Morrison, M.D., C.M. (Edin.).

(Notes based on an analysis of 245 cases.)

This paper is based on an analysis of the 245 cases of eclampsia which have occurred at the Women's Hospital, Melbourne, in the years 1900–1910, inclusive. During that period I have been one of the four honorary obstetric surgeons.

The 245 were all true eclampsia. Several cases where convulsions were due to epilepsy or hysteria have not been introduced.

At first I considered the advisability of only recording the cases directly under my care. I have, however, included the cases under my colleagues as giving the report much more statistical value.

For the 11 years, there were admitted 16,087 patients. Omitting all patients under 4 months pregnant, we have 14,717 cases on which to base statistics.

Year.	Cases.	Eclamptics.	Proportion of Eclamptics to Admissions.	Died.	Per cent. Deaths to total Eclamptic cases.
1900 1910 ...	14,717	245	1 in 60	61	25

The mortality has varied greatly in different years, being—
8 out of 14 or 57 per cent. in 1901, and only
2 ,, 26 ,, 7 ,, 1908.

Months of Occurrence.

Regarding the months, I find that—

131 cases occurred in the 5 cold months—May–September; and
114 ,, ,, 7 warm ,, October–April.

Nothing of any value is to be gleaned from the months when fatal cases occurred.

I have considered we were more prone to get cases, not only during winter months, but after a sudden change of weather from heat to cold.

Age.

The average age of those who recovered	22½
,, ,, ,, died...	26½
Youngest to recover ... aged	14
,, die ... ... ,, ...	15
Oldest to recover ... ... ,, ...	36
,, die ... ... ,, ...	42

The average age in fatal cases is thus higher (by 26½–22½) than the average age of those who recovered.

No. of Pregnancy.

Cases—		*Died—*	
Primiparæ	172	Primiparæ	40
Multiparæ	73	Multiparæ	21

This shows the preponderance of primiparæ as regards occurrence and of multiparæ as regards mortality, viz. :—

Occurrence—		*Mortality—*	
Primiparæ ...	70 per cent.	Primiparæ ...	23 per cent.
Multiparæ ...	30 ,,	Multiparæ ...	30 ,,

Period of Pregnancy.

This is shown by the table :—

Full time.	8 months.	7 months.	6 months.	5 months.	(?) or not stated.	Total.
101	40	24	8	3	69	245

Excluding the 69 "? or not stated," we have 176 cases, of which 141, or 80 per cent., were in the last month of pregnancy.

Eleven of the cases were from 5 to 6½ months pregnant, and of these 3 died.

In the journal of the Obstetrical Society of Paris, March, 1911, it is pointed out that eclampsia during the early months of gestation is unknown in primiparæ, and only occurs in multiparæ who have long-standing and advanced lesion of liver or kidney.

Of the 11 cases :—

3 were primiparæ.
8 ,, multiparæ.

Three cases were only 5 months pregnant, viz. :—

1. Aged 42 vi para—died.
2. ,, 30 i ,, —recovered.
3. ,, 42 vii ,, ,,

The primiparæ aged 30 was admitted after a fit. She had general œdema and a history given of frequent vomiting and occipital headache. She had three more fits, and then labour was induced with a bougie and plugged vagina. She recovered.

Premonitory Symptoms.

In their order of frequency, the commonest symptoms were :—

1. Œdema.
2. Headache.
3. Epigastric pain.
4. Visual disturbances.

Urine.

No albumen was found in 18 of the cases on admission, and all the 18 recovered.

Of the 18, one child only was still-born.

Ante	2	18
Intra	5	
Post	11	

Of the 18, only 4 had any premonitory symptoms :—

1—headache only;
1—epigastric pain only;
2—visual, œdema, epigastric.

Onset of Fits.

In many cases it was difficult, nay, impossible, to determine whether patient was actually in labour when the first fit occurred. I find that :—

Ante	71	245
Intra	84	
Ante or Intra	18	
Post	61	
Not stated or (?)	11	

In 173 cases in which the convulsions began before or during labour they continued after the third stage in 52 cases—30 per cent.

The latest cases of post-partum onset were as follow :—

1. *Primipara*, aged 31, full time. Specific history. Forceps case. Living child. No albumen, and ran a normal puerperium for a week. Without any prodromes she suddenly started fits on eighth day, and had 10 typical convulsions. Urine tested on seventh day gave no albumen, but albumen was found after fits began.
2. *Secundipara*, aged 27, 8 months pregnant. Admitted with history of having had four fits before admission, and had two in the hospital. She was treated with aperients, morphia $\frac{1}{2}$ gr., vapour bath, &c. At the end of a week she was put on thyroid extract, 5 grs., four times daily. Went out by her own request in twelve days. *Re-admitted* a fortnight later in labour. Mere trace of albumen. Face presentation. Easy labour. Living child. On the *tenth* day she had a severe epileptiform fit. I am aware that convulsions, occurring so late in puerperium, are generally attributed to some cause other than eclampsia. This, however, I look upon as a genuine case, the latest onset I have met with. The history of eclampsia with her first child, the œdema, vomiting, and albumen, and the convulsions a month before labour came on in this pregnancy, are, to me, sufficient grounds for deciding the convulsion on the tenth as eclamptic.

Here it may not be out of place to draw attention to the value of thyroid extract in the toxæmias of pregnancy. Begin with small doses, such as grains 2, thrice daily, and increase to 5 grains four times a day.

No. of Fits.

Maximum number in patient to recover	40
Minimum ,, ,, die	1

Mortality.

The time of the onset of fits affects the mortality. I find that :—

Ante	34 per cent. died.
Intra	21 ,, ,,
Post-partum	9 ,, ,,

The total maternal Mortality—25 per cent.
The child was born alive in 139 cases.
Still-born in 79 ,,
Not stated in 29 ,,

In the fatal maternal cases the child was still-born in 70 per cent.

Re-admissions.

Three cases admitted for eclampsia underwent routine temporising treatment, were discharged improved, and re-admitted later on, either in labour or because of further fits. The three mothers recovered, but two of them had still-born children.

Multiple Pregnancy.

Twins occurred ten times }
Triplets occurred once } in 245 cases—nearly 5 per cent.

(The average in normal cases is under 1 per cent.)

This accords with the known fact that multiple pregnancy predisposes to eclampsia.

Treatment.

I will shortly give the treatment I would adopt in actual eclampsia. The treatment of threatened eclampsia will not be considered.

I take it that we must look upon eclampsia as a toxæmia caused by the absorption of certain products and ferments from the placenta.

It must be our endeavour to assist the elimination of the toxins, and, at the same time, to try to control the convulsions.

Elimination of toxins is assisted by washing out stomach and acting on excretory organs.

The patient is put to bed, a gag or spoon at hand to prevent biting tongue, vulva shaved and washed, catheter passed and urine put aside for examination, *stomach* washed out until clear return, and then half a pint with 2 ounce mag. sulph. left in. *Rectum* emptied by ordinary enema, and, if result unsatisfactory, give a high saline wash-out, or a high enema of—

Mag. sulph	1 oz.
Castor oil	1 oz.
Glycerine	2 oz.
Aqua	to a pint.

The skin is acted upon by putting patient in a hot pack for about an hour, and, at the same time, giving her plenty of fluid—

By mouth if she can swallow,
By rectum, if not too restless,
Under skin, preferably submammary; or
Intra-venously.

There is a slight risk of overloading her with fluid, but that risk can be avoided by never giving fluid intra-venously except after venesection, and then not more than two pints. When given under the skin, the rapidity of absorption must be noted. The fluid used should be normal saline, and I prefer to add 1 drachm of sod. acetate to each pint, especially if morphia has been used. The acetate is diuretic. The continuous saline by the rectum is most suitable for puerperal cases.

A *poultice* over the kidneys seems to assist diuresis.

Venesection is done in all cases where the blood pressure is high and the patient full-blooded or cyanosed. It is especially valuable in cases without œdema, and with a hard, wiry, rapid pulse. Owing to the increased coagulability of the blood it is sometimes difficult to bleed to more than a few ounces, but I prefer to get 16–20 oz. Then give two pints of normal saline solution into vein.

Oxygen is invaluable, is given in practically every case and with the greatest benefit.

Control of the convulsions must be considered.

It is important to try to lessen the irritability of the nerve centres by such a drug as morphia. In earlier days I used morphia in large doses and oftener than I do now. If the fits are occurring at all frequently I give ½ grain, and very seldom give any more, though occasionally I order perhaps another ¼ grain in 4 hours. It undoubtedly checks elimination and probably affects the child. If the urinary secretion be very scanty I now never use morphia.

Chloroform should be given if any examination has to be made or any treatment undertaken. The reflexes are so active that fits are often induced by mere vaginal or rectal manipulation. If, after employing general and medicinal remedies, the convulsions are not under control or the patient's condition shows no improvement, we must consider the advisability of inducing labour.

Far and away the most difficult cases for decision as to treatment are those in which the *patient is not in labour. When* to induce labour, if at all, and *when* to rapidly empty the uterus, if at all, are the two most debatable and unsatisfactory problems in all obstetrics. Before deciding as to treatment in a case of N.I.L., one must consider the condition of the fœtus. Should it have died, one would be more inclined to continue temporising measures. The prognosis as regards the mother is favourably affected by the death of the fœtus, and it frequently happens that fœtal death means the cessation of convulsions.

A few years ago, I invariably followed the teaching of those who advocate temporising measures: "Attend to the convulsions, and the labour will take care of itself." Whilst in mild cases I still follow that dictum, I now, in severe cases, interfere earlier than I formerly did. The fœtal mortality is so extremely high in eclamptics, not in labour, that, if we consider the full-time unborn babe to have an equal claim with the mother, we simply must deliver it as quickly as possible.

If the temporising treatment controls the convulsions, do not interfere with labour unless her general condition becomes more unsatisfactory—when you must induce labour by dilating the cervix with Hegars, putting in bougie, and packing cervix and vagina. Should her condition be very serious, one must not be content to induce labour, but may have to empty the uterus rapidly. I have not yet had a case of eclampsia which I considered suitable for ordinary Cæsarean section. Bossi's dilater was used by me on two occasions some years ago in eclamptics where the cervix was not taken up.

Hysterotomy is rapid, and not likely to cause serious injuries or hæmorrhage if the bladder be well separated. The greatest difficulty would be in full-time eclamptic primipara with fœtal head filling the pelvic brim. In a vast majority of cases labour ensues shortly after the onset of convulsions. It is certain that eclamptics have more powerful uterine contraction than other patients, consequently, when the pains do come on, labour is generally fairly rapid.

Should the patient be in labour, leave her alone if the pains are strong and good progress is being made. If not, then expedite the *first stage* by manual dilatation or De Ribes' bag, and the *second* stage by forceps. With patient lightly under chloroform I frequently have put on forceps before the os was fully dilated and used them as dilaters as well as tractors. Should the cervix be rigid I do not hesitate to incise it freely, if time be an important factor for mother or child. Should she be primipara, with anything like a small outlet, I do episiotomy. One always likes to see a fairly free hæmorrhage post-partum.

There are certain drugs which at one time or another were greatly in vogue in treating eclampsia, and are even now vaunted by many, but all have been discarded by me.

Croton oil.—Useless in dose (1 m.) ordinarily prescribed, and larger doses, even up to 6 m. or 10 m., are too uncertain.

Pilocarpin.—Not used for many years because of risk of inducing pulmonary œdema and œdema of glottis.

Veratrum viride, so largely used in America, has never been found satisfactory. It certainly reduces blood pressure, but only very temporarily, and in large doses affects the child.

Chloral.—Discarded because often difficult to administer; uncertain in action, and depressent to the heart.

SUMMARY OF TREATMENT.

1. *If not in labour*—
   (*a*) Mild case—temporise—Control the convulsions;
   Eliminate toxins.
   (*b*) Severe case—Induce labour early;
   (*c*) And the more severe the case the more rapidly must the uterus be emptied.

2. *If in labour*—
   (*a*) Leave alone if the pains are strong, and there is good progress.
   (*b*) If delay and condition serious, then expedite.

3. *Post-partum*—
   Elimination.
   Control of convulsions.

NOTES ON ELEVEN CASES OF PUERPERAL ECLAMPSIA.

F. C. STEVENSON, L.R.C.P. & S. (Irel.), Moss Vale.

I WISH to bring under your notice to-day a series of eleven cases of puerperal eclampsia which have occurred in my practice during the last twenty-seven years.

I trust the history of these cases may prove of some interest to you, for the reason that in most instances they have had to be treated promptly, single-handed, in remote country districts, without the help of even a trained nurse, thus differing materially from the larger series of cases published, occurring for the most part in large institutions, with plenty of highly-trained assistance available.

In three out of eleven cases I was fortunate in being able to secure the help of other medical men, and these three cases all survived. In the remaining eight cases I was alone, and had to do the best I could single-handed, and of the eight cases six recovered and two died; so that out of the total eleven cases there were nine recoveries and two deaths.

The first case I lost was in Moss Vale in 1896. The patient was a multipara at nearly full term, who sent for me suffering from severe gall-stone colic; she was deeply jaundiced. I gave her a hypodermic of morphia, and on the next day she was much better. On the following day I was again summoned at 3 a.m., and found her in a state of coma, with frequently recurring uræmic convulsions. While I was with her, Nature effectually emptied the uterus of all its contents, and within two hours of this spontaneous delivery she died.

The second case I lost was in June, 1910. The patient in this instance was quite a young woman, and a primapara. She lived 20 miles away, and on my arrival the child had been born some time, and the third stage completed. She had a running uncountable pulse, was cold and clammy, and had severe convulsions, recurring every few minutes. She died within three hours of my arrival.

In both cases the women were delivered naturally without any interference on my part, and died so soon after the completion of labour that I cannot imagine any treatment could have proved of benefit.

In the remaining nine cases, the treatment adopted has been uniformally the same in principle, namely, to administer chloroform sufficient to procure deep anæsthesia, then to dilate the os digitally and as rapidly as possible, and deliver by podalic version. This procedure has usually been accomplished, in time varying from twenty minutes to one hour. After delivery, if the fits continue, morphia has been administered in quarter-grain doses to control the convulsions, the bowels freely moved with large doses of Epsom salts when practicable, and nitro-glycerine given in 2 minim doses every four hours hypodermically while the patient was unconscious, and subsequently by the mouth.

In Case II of the series, the patient lived in the bush, 15 miles from Scone, where I was then practising. In consequence of the people failing to inform me of the nature of the case, or of her condition, I found myself confronted with a primipara in a state of coma, and having convulsions at frequent intervals 15 miles from home, without chloroform or midwifery-bag. To save time I placed her in my buggy, and had, what was to me, the unique experience of driving a patient with eclamptic convulsions for 15 miles. On arrival in the township, chloroform was given, and the patient rapidly delivered. She made an uneventful recovery.

In ten out of the eleven cases, convulsions supervened either before or during labour. In one case, the sixth, they came on after an easy normal labour. The fits were very severe and frequent, lasting four days, during which time she was unconscious; the convulsions were of the tetanic type, the facial distortion horrible, and accompanied by opisthotonos. Her recovery appeared to be unlikely. The treatment adopted was the hypodermic administration of ¼ grain morphia and 2 minims of the 1 per cent. solution of nitro-glycerine, alternately, at intervals of four hours. The urine contained albumen for three months after confinement. This patient had been previously confined twice without any complications, and

I have since attended her again in a perfectly normal confinement, with no trace of albumen in the urine either before, at, or after the time.

In passing, it may be of interest to mention that three of these cases occurred in a place called Wingello, a small village on the Main Southern line, containing about fifty inhabitants. The proportion seems an unduly large one for so small a handful of families.

In Case III of the series, which I attended in Tasmania in 1893, the presence of general anasarca and œdema of the lungs, materially added to the gravity of the case. The urine was solid on boiling, and uræmic convulsions supervened, when the patient was seven months advanced in pregnancy. The administration of chloroform was rendered difficult by her water-logged condition, and artificial respiration, and the employment of amyl nitrite, had to be resorted to to help us through. However, after delivery, there was rapid improvement, both mother and child surviving.

As regards injury to the patient consequent on the rapid dilatation and delivery, I have to record the laceration of the cervix in Case Seven and an extensive laceration of the perineum in Case Eight. In the latter case, I believe the same result would have eventuated under ordinary conditions owing to the age of the patient and the smallness and rigidity of the parts.

I may now briefly mention some methods of treatment suggested by various authorities:—

Cæsarean section is strongly recommended by some, but necessitates the requisite skill, assistance, and accessories to render it practicable.

Decapsulation of the kidney, for the same reason, scarcely commends itself to the general practitioner.

Hale White recommends bleeding as useful when the arterial tension is high, and at the same time saline infusions may be given.

Lumbar puncture also has its advocates, and among drugs which have been suggested may be mentioned:—

Morphine.
Scopolamine and morphine.
Veratrum viride, in small and frequent doses hypodermically.
Nitro-glycerin.
Also, calcium salts have been recommended, as they are abstracted from the blood in this condition.

The general impression seems to be that puerperal eclampsia is due to the presence of some toxin or toxins in the system, and on this assumption Liepmann deprecates sweating as being likely to concentrate the toxin in the blood, and thus do harm. This line of argument appears to be strictly logical.

In 1893, Schmorl demonstrated the presence of focal necroses in the portal spaces of the liver in eclamptic cases; and lately, H. Leith Murray, of Liverpool, has described a complicated toxin, which he considers responsible for these focal necroses, and the hæmorrhages in the brain, and which resembles in many of its features the poison in snake venom, and suggests the use of antivenin in eclampsia, or the pre-eclamptic state.

To summarise, if I may venture to draw conclusions from so small a number of cases as the eleven which I have attended, and brief notes of which are appended to this paper, I feel inclined to ally myself with those who advocate immediate and rapid delivery directly severe convulsions occur.

I believe that the hæmorrhage which follows the removal of the placenta is beneficial, and tends to bring about a cessation of the fits; and if the coma and convulsions continue after delivery, I would suggest the use of morphine and nitro-glycerine hypodermically in small doses at intervals of four hours alternately.

CASE I.

Mrs. B., attended in Rugely, Staffs., England, in 1884.—Primipara at full term, had uræmic convulsions at frequent intervals, in a state of coma when seen; urine loaded with albumen; no signs of labour; os not dilated.

Chloroform was administered, and the uterus completely emptied of its contents. The after treatment consisted of the administration of purgatives and bromides with chloral.

This patient made a rapid and uneventful recovery.

CASE II.

Mrs. T.B., in Scone, New South Wales, in 1889.—This patient lived in the bush, 15 miles from my house. She was in a state of coma when visited. Primipara at full term. Urine highly albuminous, and no sign of labour; in convulsions.

I placed her in my buggy, and at once drove her into the township to a house close to my residence, where I carried out the same treatment as in the previous case, with the same satisfactory result.

CASE III.

Mrs. R.H., of Longford, Tasmania, 1893.—In this case the urine was solid on boiling, and the presence of general anasarca and œdema of the lungs materially added to the gravity of the case. Uræmic convulsions supervened when seven months advanced in pregnancy. The same treatment was carried out with recovery, both mother and child surviving, the only additional treatment being the subsequent free administration of iron and digitalis.

CASE IV.

Mrs. G., of Moss Vale.—On the 19th of March, 1896, she was seized with a very severe attack of gall-stone colic, one of many similar attacks; deeply jaundiced, urine scanty, and containing much bile; in adition, she was pregnant at nearly full term. I administered a hypodermic of morphia, and left her. On the 20th the patient was better; on the 21st, I was again summoned at 3 a.m., and found the patient in a state of coma, with frequently recurring well-marked uræmic convulsions. While with her, Nature effectually emptied the uterus of all its contents. Within two hours of this spontaneous delivery, she died.

CASE V.

Mrs. F., Moss Vale, 28th December, 1897.—This case was the mildest I have seen—a lady moving in a high social scale. She had been living an indolent life, and satisfying a large appetite with full meals of highly nitrogenous food. When called to see her, she was within three weeks of full term, had one convulsion, and was in a state of semi-coma. Urine contained a considerable quantity of albumen. I gave a brisk purgative, and having secured a free action of the bowels administered antipyrin. This patient was subsequently put on a milk diet, and aperients given until full term, when she was confined without any further complications.

CASE VI.

Mrs. D., Moss Vale, 24th November, 1900.—A highly excitable neurotic woman, confined on above date at full term, rapidly and naturally. The convulsions commenced after the birth, and increased in severity, continuing on Sunday, the 25th; on the 26th, she was still in a state of complete coma, convulsions occurring every hour, with a temperature of 102; pulse, 140. I administered hypo. morph., gr. quarter, and hypo. of nitro-glycerine M (II) alternately, at intervals of four hours; on the 28th she became conscious, and had a normal temperature, and from this date she slowly recovered. Urine contained albumen for nearly three months after confinement. This patient had been previously confined twice without any complications, and I have since attended her again in a perfectly normal confinement, with no trace of albumen in the urine before, after, or at the time.

CASE VII.

Mrs. L., of Wingello, 28th March, 1902.—This patient, living 20 miles from here, primipara, had been in convulsions some hours before my arrival. When I saw her, there was no dilatation of the os. The urine, on boiling, was solid, and the convulsions were recurring at intervals of a few minutes. I gave chloroform, dilated the os digitally, and delivered rapidly by version in about half an hour, lacerating the cervix badly in so doing. I gave morph. and nitroglycerine alternately, as in the previous case. On the third day, after delivery, she became conscious and made an uninterrupted recovery.

CASE VIII.

Mrs. L.B., æt. 30, Exeter, 17th July, 1906.—I was asked to visit this patient because she was suffering from intense headache. She was a primipara, and lived 7 miles from my residence. On my arrival, I found her in a state of semi-coma, and within fifteen minutes of my arrival she had her first convulsion. She had three fits at frequent intervals, and I decided to give chloroform, dilate digitally, and deliver at once, with the assistance of the only woman available. This was an exceptionally difficult case, on account of the age of the patient, and the rigidity and smallness of the parts, as a result of which the child was still-born, and the perineum was lacerated into the rectum. The lacerated parts were brought together with sutures, and she now has a useful perineum, with complete control of the contents. The treatment was similar to that employed in previous cases, namely, morphia, nitro-glycerine, and free elimination by the bowels. She made an excellent recovery after many anxious days, and has had good health since.

CASE IX.

Mrs. S., Moss Vale, 11th January, 1910.—A big, healthy woman, multipara, and had several children without trouble, and usually without medical aid. On my arrival, I found her unconscious, and having frequent convulsions. I was fortunate in securing the help of Dr. Jones at once, who gave chloroform for me, and with little difficulty, and no injury, I delivered her rapidly of a live child.

Complete consciousness did not return for four days; otherwise she gave me no anxiety, and made an uninterrupted recovery. The same treatment being adopted as in the previous cases. The urine was nearly solid on boiling.

CASE X.

Mrs. G.G., Wingello, 8th June, 1910.—The patient, a young woman, primipara, lived over 10 miles from Moss Vale. On my arrival, I found the child born, and the third stage completed, but the mother in a parlous state—cold and clammy, with a running, uncountable pulse, and convulsions frequently recurring every few minutes. I gave a quarter-grain of morphia hypodermically, and controlled the convulsions slightly, but recognised that she was moribund. She died three hours after my arrival, apparently an utterly hopeless case from its inception.

CASE XI.

Mrs. J.D., Wingello, 30th June, 1910.—Strange to say, I was summoned to this case twenty-two days after the last fatal case, in the same place, a small village containing about fifty inhabitants. This patient was also a young woman, and a primipara. She had had several convulsions before my arrival, was comatose, and the fits continuing, I followed my usual procedure at once—chloroform, digital dilation, podalic version, and after delivery, the administration of morphia and nitro-glycerine.

The urine was highly albuminous, but the albumen quickly disappeared after delivery, and she made a rapid and satisfactory recovery.

The following papers were also submitted, but owing to the absence of the authors were not read: —

1. Note on Non-Surgical Treatment of Cancer, by A. D. Leith Napier, M.D., &c.
2. Malignant Uterine Disease, by A. D. Leith Napier, M.D., &c
3. Potassium Iodide as an Antigalactogogue, by C. F. Hodgkinson, M.B., Ch.B. (Melb.), Tasmania.
4. 200 Consecutive Cœliotomies, by A. J. Nyulasy, M.R.C.S. (Eng.), Perth, W.A.

ONE THOUSAND CONSECUTIVE CONFINEMENTS IN PRIVATE PRACTICE.

By Herbert Osburn Cowen, M.D. (Glas.), Kew, Victoria.

This record comprises 700 cases, which formed the basis of a paper read at the Intercolonial Medical Conference at Hobart in 1902, and 300 cases attended since that date, the whole forming a series of 1,000 consecutive confinements. The conditions and circumstances of the work were as follows:—

1. Responsibility has been assumed for all cases in which I have been present before the delivery of the placenta.
2. No post-partum time limit has been imposed. Personally, I do not think the statutory thirty days sufficient. I have known cases in which, had death occurred six or more weeks subsequently, it would have been as clearly due to the confinement as if but one week had elapsed.
3. Miscarriages before the fifth month have not been included, though no fatality has occurred from this cause, as I consider them a factor in a somewhat different equation.
4. The cases have been taken in the course of a mixed general practice, and with very few exceptions, in spite of post-mortem work or the currency of a septic case.
5. The first 700 cases in the series occurred in a working-class neighbourhood, chiefly mining. During this period only untrained nurses and relatives were available.
6. The remaining 300 represent my practice in a good residential neighbourhood. During this period I have almost invariably had the assistance of efficiently-trained nurses.

Preliminary Considerations.

It has been said that the medical man begins his struggle with infection on laying hold of his obstetric bag. But he must begin it long before this, recognising that successful warfare depends hardly less upon preparation than upon performance. Apart altogether from the added horror of puerperal infection, pregnancy and parturition in themselves involve no small risk and strain. Even under modern conditions there still remains a small proportion of women to whom pregnancy brings improved nutrition and general health; but for the large majority it involves more or less impairment of function, digestive, hæmapoietic, nervous, and mechanical. There results loss of individual resisting power and of tissue vitality. Deficient innervation, for example, and inefficient muscular contractility increase the liability to post-partum hæmorrhage. The resulting diminution of systemic resistance to microbial invasion and the greatly enhanced activity of the absorbents seriously increase the liability to generalised infection. And over and above its special significance, albuminuria, with the cellular degradation to which it gives rise, has an important bearing in this direction. In short, from the date of his engagement to attend a confinement everything that concerns the functional and organic health of his patient must concern the practitioner who desires to conduct her with the minimum of risk through her coming trial.

The Obstetric Bag.

It has always seemed to me that quite an unnecessary amount of abusive criticism has been directed against the obstetric bag. Various measures have been suggested for its reformation, and in vain; its abolition is now demanded. The so-called aseptic detachable lining is a delusion, and may be in a very real sense a snare. As it is impossible to keep such a lining aseptic, it cannot be looked upon as a defence, and after two or three boilings it becomes utterly lost to all sense of size and proportion. The bag should certainly be clean, but it is the man who must be aseptic. The principle which so much insistence upon the character of the bag is apt to make us lose sight of is that the only safety in obstetric work lies in personal and instrumental sterilisation for each individual case. The obstetric bag should be kept strictly for primary obstetric work, and it should be much larger than that in common use, in order to carry in washable coverings an apron and the necessary instruments and appliances.

Preparation of Patient and Physician.

After reaching the sick room, and noting the general condition of the patient, the medical attendant's first duty is the careful sterilisation of his hands. I need not go into detail as to this, but need only say that it should be as conscientious and thorough as for any surgical undertaking. If, owing to previous septic contacts, he is in doubt of its completeness, sterilised rubber gloves should be worn. The nurse will, in the meantime, have been engaged in the careful preparation of the skin at and around the vaginal orifice, and the combined abdominal and vaginal examination of the uterus may now be undertaken.

Vaginal examination must only be performed after thorough preparation of the physician's hands and the careful separation of the labia minora, in order that the examining finger may not carry infective organisms from the external genitalia to the upper part of the vagina. In a large proportion of multiparous cases it may be entirely dispensed with, and in primiparous cases where the head is well advanced. But if we keep before us as our ideal not to make more than one vaginal examination, and not to make that if we can avoid it, we can so safeguard the procedure as not only not to increase, but actually to diminish, the risk to mother and child.

Much as we may learn from abdominal palpation as to the position of the child, and even as to the progress of labour, there remain points upon which vaginal examination only can inform us, such as the condition of the cervix, the descent of the anterior lip—which not infrequently prolongs labour and renders it more painful—and the exact bearing of coccygeal abnormalities; for the early recognition, too, of prolapse of the cord, upon which the life of the child may turn, we are entirely dependent on vaginal examination.

I am interested to note that, in speaking of abdominal palpation, Berry Hart says:—"The method is not so suitable for private practice, but there the reduction of internal examination to a minimum, with the use of gloves, should give results as good, and probably more sure." Needless to say, the casual "trying of the pains" by the nurse must be absolutely prohibited. For the rest, in the conduct of a normal case of labour it is well for us to bear in mind that it is a natural and not a morbid process with which we have to deal, and that nature has provided safeguards for the parturient woman in the way of secretion, contraction, and direction of flow. It is for us to do our utmost to avoid contaminating those secretions or unnecessarily interfering with those processes.

Presentations, &c.

In the series of 1,000 cases, 287 were primiparæ; of the presentations, 975 were cephalic. Of these, 53 were noted as being occipito-posterior; but this cannot be taken as the full number of such positions, as some may have rotated before my arrival. My personal experience of occipito-posterior positions leads me to question the approximate accuracy of Dr. West's estimate that not more than 4 per cent. terminate with the face to the pubis. Of my last thirty cases of occipito-posterior positions, I find that no less than ten terminated in this way, and delivery in these was by no means always difficult. But in view of the relative frequency of the occipito-posterior position, and the greater difficulty and delay which it frequently entails, the question of its correction seems to me to be most important. When the head remains in this position my present practice is to rotate the head with the left hand in the uterus and the right hand over the fundus. Having thus rotated, I hold the head in position until the blades of the forceps are accurately applied. More tentative efforts at digital rotation and the application of the forceps, without being absolutely certain that the corrected position was maintained, almost without exception ended in disappointment. The subsequent difficulty in delivery, with its attendant traumatism of the materal soft parts, being explained by the appearance of the vertex in its original faulty position. I have not attempted rotation with the forceps on, notwithstanding Smellie's "great joy," as I do not know what damage the blades may be doing in positions in which I am not accustomed to handle them. Upon no point in the management of difficult labour has my individual experience thrown such clear and definite light as upon this. I had thought and read much about it, and keenly realised its difficulties, and theoretically at least knew the way out. But my efforts were tentative and half-hearted, and too easily influenced by the anxiety of the moment. Suddenly, however, two consecutive cases crystallised my thinking and theory into a practical procedure. Both were second cases, and in both the first labour had been very difficult. In the first, which was a case of my own, I had the assistance of a colleague of great experience. The head remained high in the occipito-posterior position, and we both tried unsuccessfully to deliver with forceps, and failed to move the head at all, owing to the repeated slipping of the blades. I then rotated the head and applied the forceps with the left hand in the uterus as detailed above, and delivered a dead and mutilated child without undue difficulty. The second was a case in which a fellow practitioner asked me to assist him. He told me that the first confinement had been most difficult and protracted, and that the child had died during delivery. He had on this occasion made repeated attempts at the accurate application of the forceps, and had failed. In this case I did not attempt to apply the blades until I had rotated and retained the head in position. The result was entirely satisfactory, and delivery of a living unmutilated child was effected without difficulty. I lay no claim, of course, to originality for this procedure; I only regret that I had not boldly adopted it long before, and that it is not more frequently resorted to by practising obstetricians.

There were four face and brow presentations and two of the whole vertex, in which neither brow nor occiput showed any tendency to engage. I can find little as to this condition in the text-books, but I am quite clear that these were two of the most difficult cases to deliver which have occurred in my practice. Both were large, square-headed children; in both the head remained high, and the forceps showed an inveterate tendency to slip. Ultimately version was performed, and both children were born dead. Looking back, I cannot but regard the failure with the forceps as fortunate, since delivery by that means must have involved serious injury to the maternal soft parts.

There were twenty-one presentations of the breech, one of the funis, two mixed (one arm and head and one hand and breech), and two transverse. Of three cases of placenta prævia two were partial and one central, the latter accounting for the only maternal death in the series. Of eight cases of twins two were premature, and in two the second child was still-born.

Accidents.

Of three cases of eclampsia, one occurred during labour, one six hours, and one sixty hours subsequently. The two post-partem cases were very severe, but one has a much freer hand in treatment when delivery has not to be considered. All three mothers recovered. These cases occurred in my first series. In the second series I have been confronted with a proportion of cases of pronounced albuminuria with dropsy, but treatment has been instituted earlier, control has been closer and more continuous, and in no case has eclampsia developed. I do not propose to discuss the much-debated question of the causation and treatment of eclampsia, but my later experience has confirmed my preference for its preventive treatment. Purgation, diuresis, milk diet, and later on sedatives, have, as it seems to me, saved me much anxiety, while they have at the same time restricted my experience of eclampsia.

In 975 head presentations forceps were applied in 164, practically an average of 1 in 6. Of 287 primiparæ, 96 were forceps cases, an average of 1 in 3. I do not find any marked difference in the ratio as between my two series of cases, one representing practice in a working-class and the other in a good middle-class neighbourhood. My later series shows a slightly diminished ratio for primiparous in a slightly increased ratio for multiparous cases, indicating, it may be, a greater relative frequency of uterine inertia; but the numbers are not large enough to establish anything definite on this point. As far as I am able to gauge it, my tendency is to leave more to the natural efforts of the primiparæ, and to assist those of the less vigorous multiparæ in the later stages of delivery. Milne Murray's aphorism is excellent: "Only apply the forceps when the danger of delivery is less than that of delay." But what a rare amount of wisdom and experience its true interpretation requires. Of this I am persuaded that the too early and too frequent use of the forceps is an important factor in the maintenance of the high puerperal mortality and morbidity rates. Important as trauma is from the point of view of mortality, it is even more so from that of morbidity. Broadly, I think it may be said that the morbidity of the puerperium is more largely determined by the traumatism produced during the second stage of labour, and its mortality by the complications of the third stage.

The Management of the Third Stage.

This, more than anything else, marks the dividing line between safe and dangerous midwifery. In it lies the key to the prevention of post-partum hæmorrhage, and in the great majority of cases of puerperal sepsis also. Antiseptic precautions may carry the accoucheur safely through many an error in the use of the forceps, but they will not save him from the consequence of want of thought and want of patience in the delivery of the placenta. So far as I am aware, it is only within the last few years that the necessity for a more rational and deliberate treatment of the placenta has been borne in upon the profession at large. Credé's method had become a positive fetish, and comprised all that was worth knowing about the third stage of labour. It was indeed all that I carried away of the teaching in my own student days on the subject. And yet, practised as it has so generally been without limitation as to time and force, I take it to have been the most important factor in the maintenance of the puerperal death-rate. It is not, I think, generally remembered that shortly before his death Credé recommended a lapse of thirty minutes before his method was to be attempted. My own practice is to follow down the uterus after the birth of the head, and then, with the hand over the uterus, to watch rather than control it for twenty minutes. I am sure I did harm in my earlier cases by trying to maintain contraction of the uterus at this stage; it is not Nature's way; it is goading the jaded steed, which only asks rest and it will do all that is required. I have not seen hour-glass contraction since I abandoned this procedure.

After a few pains the uterus will be found to rise above the pubis, this signifying, according to Professor Dyers, that separation of the placenta has been effected. Then, during a pain, with the whole hand square above the fundus, I follow down the uterus, and it is not until I have repeated this manœuvre without success that I attempt expression by Credé's method. In more than 95 per cent. of my cases nothing further has been required. If, however, after the lapse of half an hour, further attempts at expression give the impression of organic immobility, which one soon comes to recognise, I do not hesitate to remove the placenta manually. I find that this has been done thirty-eight times in all. With careful preparation of the hand and forearm, I have found the procedure both safe and satisfactory, and I am convinced that the warnings of the older text-books, as to the grave nature of the proceeding, have often tempted the attendant to employ force sufficient to detach the main body of the placenta from an adherent portion. It is only in cases in which forcible expression has been employed that I have found difficulty with the membranes. I then support the placenta on the palm of the hand, and, waiting for relaxation of the uterus, use gentle traction in the axis of the canal, always stopping short at the suggestion of a "give." I prefer this to the method of twisting the membranes so commonly recommended, as this seems to reinforce the stronger as against the weaker portions, and so to increase the liability to tear.

As a matter of routine, the placenta and membranes are examined, and if I conclude there has been a retention of a portion I go in search of it at once. In cases where early escape of the liq. amnii suggests that the membranes are unusually friable, additional care is necessary in the removal of the placenta. In such a case I have known the placenta to appear stripped entirely of membranes by its own weight.

The Post-partum Pulse-rate.

On the conclusion of labour there occurs, in a fair proportion of cases, a phenomenon, to which it seems to me sufficient attention has not been given. I refer to a marked fall in the pulse-rate. One text-book speaks of this as beginning eight to forty-eight hours after labour; another, vaguely, has been most marked on the second or third day. In a series of 100 confinements I have notes of its occurrence in nine cases. Of these three were in first cases, and gave the following counts:—

1. Ante-partum pulse-rate not recorded, but next morning pulse was 50, counted to the full minute, and on the third day it was 58.
2. Ante-partum pulse-rate, 65 to 70; fell to 44 within quarter of an hour after the expulsion of the placenta.
3. Very tedious case; pulse 64 before putting on forceps under chloroform; placenta was expelled spontaneously, and pulse fell to 53.

Two were second cases, one a third, two fourth, and one sixth. In the last the ante-partum pulse-rate was 66, and there was great distension of the uterus, both placenta and child being very large; six hours after the pulse was 45, and on the second day 60. They all gave counts of 53 to 44. In one only was the lowest count noted as occurring thirty hours after, and in the remaining eight it was after the expulsion of the placenta. These are cases which almost invariably make good recoveries, and it may be inferred from this that there is nothing pathological in the phenomenon. The explanations offered in the text-books that "it is due to the mental and physical rest which follow delivery," and the sudden diminution in the amount of labour put upon the heart, &c., do not at all satisfy me. These are common to 95 per cent. of confinements, but the fall in the pulse-rate occurs in but a small percentage. I am not prepared to define the complete significance of the phenomenon, but I have a suggestion to make as to its occurrence. I premise that the parturient woman has a varying physiological amount of blood to lose. When this is exceeded the pulse rises, and much in proportion to the excess, the heart being spurred to supply the wants of the economy with a diminishing quantity and quality of blood. On the other hand, when less than the usual amount of blood is lost, and this has been true of all the cases I have observed, the heart has more blood for distribution than is actually required, and beats more slowly until the status quo is regained. It may be, too, that the infrequency of septic complications in these cases is explained by the engorgement of the circulation reducing absorption to a minimum.

Repair of Lacerations.

Experience has convinced me of the folly of pursuing the policy of the open-door with regard to lacerations of the perinæum. If extensive, they should be repaired in the dorsal position, and deep and accurate apposition obtained. I am at a loss to explain Nature's apparent indifference to the integrity of the perinæum, even in normal cases, seeing that it constitutes such an important safeguard against puerperal infection.

If for any reason there has been recourse to forceps extraction before the full dilatation of the cervix, it is advisable to explore the upper reaches of the vagina and the cervix, as rupture into the posterior fornix is known to occur as a result of instrumental delivery in such cases, while the perinæum itself remains intact. In normal cases, however, and even in the large majority of difficult cases, post-partum interference is to be strongly deprecated. For this reason routine post-partum douching has been abandoned. In my opinion, vaginal douching post-partum is worse than useless as a prophylactic, and it is only called for as a preliminary to intra-uterine irrigation. For such a procedure only a fountain douche should be used, because of its one-way flow, the domestic Higginson, with its two-way suction being, to my mind, a dirty and dangerous instrument, over which one can exercise no efficient control.

Puerperal Sepsis.

Our attitude towards septic infections has so radically altered during the past twenty years that it is hard for us to realise the point of view from which it was then regarded. As late as June, 1894, the *Lancet* reports a discussion at the Royal Academy of Medicine of Ireland on the ætiology, prevention, and treatment of puerperal fever or septicæmia. It was introduced by a gynecologist of world-wide reputation, and in the course of his remarks he spoke of the epidemic diffusion and local prevalence of puerperal septicæmia due to air-borne "germs" much as if he were discussing a disease of the nature of measles or scarlet-fever. I venture to say that no ordinary practitioner to-day, who makes claim to the modern point of view, would for a moment think of sheltering behind a proposition such as this.

To-day we acknowledge, not without fear and trembling, that sepsis occurring in a healthy woman during the puerperium, is due to some error on the part of her attendants.

But not only has the old complicated view of these conditions failed to lay the blame on the right shoulders, it has also created a paralysing amount of needless alarm. I have had, I regret to say, several cases of sepsis in my series of 1,000, the most serious of which I reported at some length to the Victorian Medical Society; but they have all, without exception, yielded to treatment. I attribute this largely to the simplification of the situation in my own mind. For all practical purposes, puerperal fever, so-called, has resolved itself into secondary infection of retained membranes, placenta or clot, or of vaginal or uterine laceration.

It has been usual to distinguish at this point between sapræmia, or septic intoxication, and septicæmia, or septic infection. It is now generally admitted that this is a distinction of degrees and of duration only. The attack of pathogenic organisms upon dead tissue-producing toxæmia may, at any moment, develop into a general invasion producing septicæmia. It is more correct, therefore, and it is certainly safer clinically, to record the so-called sapræmia, as a stage on the road towards a general septic infection, and to treat the case from the outset as having all the inherent possibilities of the latter. Fœtor of the lochia is not regarded as evidence of puerperal sepsis. In some of the most virulent cases of streptococcic infection it may be entirely absent. It is evidence of decomposition of dead tissue, but tells us nothing as to systemic invasion; in fact, given other evidence of general infection, I should regard marked fœtor of the lochia as a favourable symptom. It suggests local rather than general bacterial activity, and it promises a satisfactory response to energetic local treatment. Where it is possible, a bacteriological examination should be made of the uterine contents, and, if performed early, this may give invaluable indications for treatment. In the later stages of puerperal sepsis mixed infections so confuse the issue that the indications for treatment are much less definite.

Treatment.

Given the presence of infective organisms in the parturient canal during the puerperium, and everything favours their multiplication and distribution. This is no time for hiding one's head in the shifting sand of conjecture. Above all other explanations, let us banish the ever-handy "influenza" from our consideration of the earlier, and "typhoid" from that of the later stages of puerperal sepsis. Every hour is of importance. Acute immediate general infection, presumably streptococcal, is comparatively rare, and the development of puerperal sepsis may frequently be inferred before a general microbial distribution has occurred. Such inference should spur us to immediate action. If we have been quite sure of the integrity of the membranes and placenta, it will be sufficient in the first instance to douche the vagina, and subsequently the interior of the uterus, and to deal with such breaches of surface as our examination reveals. This will remove the uterine debris, destroy organisms which have not penetrated the decidua, and, according to Pryor, if performed within twelve hours of the onset of the first evidences of infection, will effect a cure. If it does not, and especially if there has been any doubt about the placenta, my own practice has been to explore the interior of the uterus under an anæsthetic, and to remove adherent tissue and debris with a blunt flushing curette; thereafter to swab out the uterine interior with an antiseptic strongly germicidal, but not escharotic; to irrigate with a normal saline or very dilute antiseptic solution; and then pack the cavity with 10 per cent. iodoform gauze. In the limited field of private practice I have not found it necessary to employ the more radical procedure of the cul de sac operation so strongly recommended by Pryor. Such is my attitude towards the much debated question of primary curettage in cases of puerperal sepsis. It has given me most satisfactory results, and carried out carefully, and with a rigid regard for asepsis, has proved itself a sound and safe procedure. As to the wisdom of curetting when secondary endometritis has become established, I am not prepared to dogmatise. Theoretically, as it seems to me, such a condition would be most scientifically attacked from the side of the blood stream, with such reinforcements as bacteriology could suggest in the form of personal vaccines and sera, and by free drainage of the uterus. In the case of direct virulent streptococcic infection, in which there is no question of retained secundines, curettage is not indicated; but these are cases the true nature of which can only be determined by bacteriological examination, and in which it is reasonable to expect good results from antistreptococcic serum.

Results.

Fœtal Mortality.

In all, twenty-six children were born dead; but of these, eleven had been dead for some time, as indicated by pealing and maceration; one was hydrocephalic, two were the second of twins, and two were born dead before my arrival. This leaves ten fœtal deaths directly attributable to difficult and retarded delivery under my own control. It is difficult, at this distance, to get statistics as to fœtal mortality in this restricted sense, and I do not know how these results compare with others.

Maternal Mortality.

There was one maternal death due to central placenta prævia. In view of the high mortality in such cases under the best possible conditions and treatment, this can hardly be considered a preventable death.

In Conclusion.

I freely admit that I might have been equally careful and yet less fortunate. In not a few cases, at the termination of labour, the condition of the mother was such as to cause the greatest anxiety from shock or hæmorrhage or heart failure, singly or combined, but in all the balance ultimately turned in favour of recovery. I have had, too, especially in the first series, a proportion of cases of puerperal morbidity due to sepsis, some of them alarming, but all have yielded to treatment. I recognise that in the latter class, as well as in the former, the favourable issue was largely beyond my control.

I am none the less glad to be able to present this Section a series of 1,000 consecutive confinements without a maternal death from preventable cause.

DISCUSSION ON DR. COWAN'S PAPER.

Dr. Ludowici (Sydney) said that he must congratulate Dr. Cowen on his paper, for not only had he dealt ably with a most important subject, but had added considerably to its value by the addition of statistics, which always involve a considerable amount of labour.

He entirely agreed as to the necessity of recognising the position of the head as early as possible; as to the frequency of the occipito-posterior condition, in investigations of over 3,000 cases of labour in district practice of the Women's Hospital, Sydney, he found that the percentage of occipito-posterior cases was 4·7 per cent.; this is very different from that given by Fothergill and Whitridge Williams—23 per cent.

His own custom was not to leave a case until thoroughly satisfied about the position; and if abdominal and the ordinary vaginal examination were not sufficient, he did not hesitate to administer chloroform so as to facilitate palpation of the head and its various landmarks. In treatment of persistent cases, the object should be to rotate the head. Whilst this was usually easy, care should be taken to rotate the body at the same time, with one hand upon the abdomen and by posturing. Should this not be successful (as often very little fluid surrounds the fœtus), the hand already in the vagina is moved up past the head, and the shoulder pushed in the desired direction. Such a precaution will prevent the head returning to its old position whilst forceps are being applied. If, as sometimes happens during the operation, the head has reverted to its old position, you will suspect it by finding that the forceps do not lock properly. Forceps once applied, the head is brought down the canal by gentle pulling and the help of the uterine contractions, not forgetting to keep the patient in a suitable position. If there be any difficulty in delivering, owing to the passages not having sufficiently dilated, the forceps are removed, and it will be found after a few hours that the patient delivers herself, or that delivery can easily be accomplished by a re-application of the forceps, thus avoiding all the risk from premature delivery through a canal not properly dilated.

Dr. Thomas Murphy congratulated Dr. Cowen on his results. He, however, could not agree that every case of puerperal sepsis was due to some error on the part of the obstetrician. He had recently operated on a case of general peritonitis caused by rupture of a pus tube in a woman pregnant two and a half months, the pregnancy being undisturbed before and after the operation. If this case had gone on to full time, or if she had miscarried and the tube had ruptured at the same time, undeserved blame might be attached to the accoucheur, and he placed this case on record to prove that the obstetrician might be quite blameless in a case of apparently ordinary puerperal sepsis.

INCREASE OF CANCER IN WOMEN.

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Abstract—

(*a*) General statistics—British, European, American, Japanese, Australasian.

(*b*) Special statistics—cancer in women—
Increase uniform, world-wide, steadily progressive.

(*c*) Causes of cancer. Heredity. Race.
"Cancer-houses": Climate, soil, altitude, diet.
Influence of irritation as cause.

(*d*) Parasitic nature of cancer—dependence of growth more upon biological character of cells than upon location.

(*e*) Education of the public upon lines similar to those applied in tuberculosis. Surgery alone reliable for cure.

(*f*) Early diagnosis essential—relation to the general practitioner—

1. Uterus. Untoward menstrual symptoms. Importance of hæmorrhage as sign. Other signs. Investigation and treatment.
2. Breast—characteristics. Importance of education of women as help towards early recognition of disease.
3. Stomach—special value of early diagnosis of ulcer and efficient treatment.

   Relation of ulcer to carcinoma.

(*g*) Conclusion—

Factors for success—instruction of the public.

Careful investigation of all cases in pre-cancerous stage by family physician.

Co-operative action best hope for reduction of cancer mortality in women.

Cancer stands to-day beside its equally virulent companion tuberculosis, slaying thousands of human beings at an incredible progressively increasing rate.

Roger Williams claims that the annual increase averages 3 per cent. to 5 per cent., and that the increase has been uniform and steadily progressive during a long succession of years.

In 1854, cancer was fatal among females at the rate of 449 per 1,000,000 living at all ages, whilst fifty years later, 1903, it had risen to 1,003 per 1,000,000.

Of each 100,000 in London since 1851, statistics show that in each decennial period the ratio has risen thus—42, 55, 68, 85, 92; and Berlin statistics for the same period show almost analogous ratios. The ratio of increase is greater in males than in females.

Williams makes the appalling observation that whilst the population of Great Britain practically doubled between 1850 and 1905, cancer mortality increased six times.

In Paris, cancer fatality rose in fifty years from 84 to 120 per 100,000; and in French towns it rose from 91 in 1890 to 106 in 1900.

During 1909, in England and Wales, 31,668 died of cancer; or for each 1,000,000 living, 749 males and 932 females, both being the highest rates on record. The increase over decennial average showed male 16 per cent., and females 6 per cent.

Turning attention briefly to other countries, we find similar increase. The Scottish Widows' Life Insurance Fund shows a remarkable steady progressive increase from 0·93 per cent. in 1844 to 6·88 per cent. in 1894, fifty years later.

The German Life Insurance Company records confirm the above, and show that, in women, cancer death-rate rose from 11·4 per cent. in 1889 to 12·9 per cent. in 1899.

In the Swedish town of Felingsbro, cancer fatality rose from 2·1 per 100,000 living in the first decennial period of the century to 118 in the last.

(1) Edwards has shown that in twenty-eight principal cities of the United States, the cancer death-rate rose constantly and generally from 48·5 per 100,000 in 1883 to 77·3 in 1907.

Cancer registration in the United States rose from 70·8 per 100,000 in 1906 to 73·1 in 1907, and 74·3 in 1908.

The Aetna Life Insurance Company, Hartford (Conn.), shows that the ratio of cancer to all deaths rose from 2·6 per cent. in 1870 to 7·3 per cent. in 1906.

(2) In Japan, 2 per cent. of patients treated in hospitals suffered from cancer, and of these 39·22 per cent. suffered from disease of the uterus or breast.

(3) Schuster, in his statistical study of carcinoma, showed the following progressive mortality per 10,000 living, of 7·14 in 1867 to 14·76 in 1907, that is, a doubled mortality in forty years.

Special interest in view of the above, bears on Australian statistics. Here, remarkable variation exists between the Australian born and the British and foreign immigrant.

Of Australian born, 58 in each 100,000 living died from cancer, as against 137 among immigrants.

Taking decennial periods 1851 to 1901, the death-rate from cancer per 100,000 living has been 14, 19, 25, 32, 45, 57. Tatham shows that cancer does not cause excessive loss of life in females until between the ages of 25 and 35. It then rises to 175 per 1,000,000, compared with 14 per 1,000,000 at the ages of 5 to 15. Onwards after the 35th year the ratios largely and steadily increase.

Coley claims that 1 woman in every 8 who reaches her thirty-fifth year dies from cancer, and that cancer has already increased to such an extent that it causes more deaths among women than does tuberculosis, the ratios being 100 to 94. He states further, that, during 10 years prior to 1861, the relative death-rate per 1,000,000 living, from tuberculosis and cancer, was 100 to 9, whilst for 10 years prior to 1900, the ratio stood 100 to 37.

There seems no doubt that during the past fifty years there has been a serious and progressive increase in cancer fatality at each successive stage of life, extending in women up to the very close.

(4) In 1854, cancer was fatal among women at the rate of 449 per 1,000,000 living at all ages; whilst fifty years later, 1903, it had risen to 1,003 per 1,000,000.

In three years, 1901–3, cancer caused death in England and Wales of 50,660 females, or 991 per 1,000,000, compared with 706 per 1,000,000 males.

This excessive mortality in females depends upon the exceptional liability of the generative and mammary organs to invasion, for if we exclude cancer of these organs, the disease is relatively most fatal to males.

Of these 50,660 females the uterus was involved in 11,714, and the breast in 8,367; that is, 20,081 women in England and Wales died in three years from malignant disease of these two organs alone.

RELATIVE Death-rates from Tuberculosis and Cancer in Women for thirty years.
(Basis, 10,000,000 of population.)

Year.	Tuberculosis.	Cancer.	Year.	Tuberculosis.	Cancer.
1881	112	37	1896	84	54
1882	115	33	1897	80	55
1883	111	37	1898	86	57
1884	112	40	1899	80	58
1885	113	35	1900	77	67
1886	105	36	1901	81	59
1887	106	43	1902	78	61
1888	101	40	1903	79	60
1889	94	45	1904	81	63
1890	97	44	1905	69	62
1891	89	50	1906	68	65
1892	89	48	1907	71	69
1893	89	49	1908	76	71
1894	87	50	1909	67	73
1895	87	52	1910	63	72

TO DR. CHENHALL (with G. H. Knibbs' compliments).—"The Australian constancy in the sum of deaths from cancer and tuberculosis is shown also in the frequency in England and Wales of the sum of these diseases. There also the death-rate for pulmonary tuberculosis has diminished, while the death-rate for cancer has increased. The following table indicates this":—

DEATHS per 1,000,000 from Tuberculosis and Cancer in England and Wales (Males), 1901 to 1909.

Year.	1901.	1902.	1903.	1904.	1905.	1906.	1907.	1908.	1909.
Pulmonary Tuberculosis.	1,487	1,456	1,427	1,458	1,347	1,356	1,341	1,310	1,270
Cancer	691	696	732	740	756	794	781	813	826
Both diseases ...	2,178	2,152	2,159	2,198	2,103	2,150	2,122	2,123	2,096

Yet we gynæcologists claim that if these cases were diagnosed sufficiently early to permit complete removal of the affected organ, practically all cases (beyond, of course, a very small mortality risk for operation) could be saved.

Better still, if, in the pre-cancerous stage, the lesions predisposing toward malignancy were recognised, especially in connection with the uterus, breast, and stomach, cancer would almost completely be prevented in these organs.

Tatham shows that, among females, out of every 1,000 fatal cases of cancer the uterus was attacked in 231, practically one-fourth of all cases. Of these, during each succeeding 10 years onwards from 25–35, the ratio rose 10, 41, 70, and so on.

(5) During six years prior to 1910, in England and Wales 104,418 females died from cancer; of each 10,000 of these 2,259 died from cancer of the uterus. Kline states that in Germany 15,000 women die annually from cancer of the uterus.

The Commonwealth Statistician, Mr. G. H. Knibbs, in a paper before this Congress, based upon statistics of the last thirty years in Australia, states that the improvement in the death-rate from tuberculosis, which is very great, is practically nullified by increase in the rate of death from cancer, inasmuch as the rates of decrease of tuberculosis and increase of cancer are sensibly the same.

Similarly, in England and Wales the death-rate from pulmonary tuberculosis has diminished, while the rate for cancer has increased.

(6) It is clear that in 40 per cent. of cases of cancer among females the generative and mammary organs are affected.

Dixon states that available statistics show conclusively that the mortality from cancer is increasing throughout the civilised world, and that in the United States it increased from 47·9 per 100,000 in 1890 to 73·1 in 1907. In the latter year there were in the country 80,000 cases of cancer and 7,000 deaths.

Guilliot found in France, by careful investigation, that the actual deaths from cancer were nearly double the number certified to having so died.

Now, it will be readily admitted that there exists among the laity an intense prejudice against the name cancer, and among family physicians an innate desire to spare the feelings of one's patients and their relatives. Hence, many secondary causes appear in certificates instead of the primary cause, cancer.

Viewed, therefore, from whatever aspect we choose, increased population, increased longevity, improved methods of diagnosis and their more constant accurate application, this enormous uniform world-wide steadily progressive increase of cancer among women during a long succession of years, is too manifest and self-evident to be doubted. The economic loss is colossal, apart altogether from the suffering and distress which is caused.

The inhabitants of rural districts seem to suffer quite as much as the dwellers in cities, of ancient cities more than of centres of industry, even allowing for the difference of constitution of the population in respect of age.

I have collected the above statistics in order to awaken special interest among the profession—and, through its members, among the public—upon the ravages of this disease, in hope that by some united effort we may add our quota of help to stay its progress.

Broadly, the great question, the cause of cancer, unfortunately still remains unsolved, and we cannot say with certainty whether it is intrinsic in origin, or enters the human body from some extrinsic or microbic source. In connection with either view, heredity deserves consideration, since the possibility of inheritance should be a special warning signal, and may lead to earlier identity of pre-cancerous conditions.

Heredity or hereditary proclivity has undoubtedly appealed to me as a clinical entity. Parents, I believe, pass on to their offspring tendencies toward reproduction of everything that these parents had acquired up to the date of their sexual congress. Statisticians may be led into errors from family records, owing to ignorance, or reticence of patients in stating that parents or others died from cancer.

The Imperial Cancer Research Fund workers were able to grow a family tree of four generations of cancer-free mice descended from a cancerous maternal ancestor. It is claimed from the evidence obtained that hereditary proclivity to cancer is so slight that it is practically a negligible quantity.

It has also been claimed that the general and local predisposition towards the disease must be acquired entirely apart from hereditary or congenital characteristics.

If this be true, then the greatest pessimist may hope that the cause, being an avoidable one, will be discovered and controlled.

Every one of us has seen instances of a number of relatives in turn becoming the subjects of cancer. So impressed am I with this fact that I cannot imagine a practitioner guided only by clinical experience coming to any other conclusion than that there is in connection with cancer something of the nature of an inheritance.

I am convinced that when younger persons are attacked by cancer there is invariably obtainable a history of family proclivity, and moreover, predisposition to development to cancer in certain organs.

Darwin has affirmed that invisible gemmules are the carriers of hereditary characters, and that they multiply by division. This is no longer a provisional hypothesis, but a well-founded theory supported (7) by histology, and the results of experimental work in heredity stand in close agreement with it. The chief characteristic of cancer is the power of originating in adult structures the fœtal modes of cell proliferation.

Such tissue proclivity handed down renders such an individual more prone than others. I am cognisant of arguments against this view of heredity in cancer, but prefer, for the present at least, to trust my clinical observation in this as in some other questions in medicine.

"Cancer-houses" I have always regarded as a myth, though some striking coincidences occur in this connection. Similarly, I do not regard cancer as an infectious or contagious disease. Cancer is, I believe, an individual disease due to transformation of healthy tissues under the influence of some causative factor of intrinsic origin.

Probably the most valuable light thrown upon the clinical aspect of this disease by the Cancer Research Fund is the proof positive that chronic irritation of living tissues, such as by pressure or rubbing of a foreign body, heat or chemicals, is an exciting cause. We all know that cancer of the floor of the mouth is very rare among women; yet in India and Ceylon, where women chew the betel nut and sleep with the plug resting on the floor of the mouth, cancer of this area is extremely common.

It is now well-known that liability to cancer depends less upon climate, soil, altitude, diet, and race than upon irritants of various kinds applied by people to the various parts of their bodies.

The logical course of procedure in search for a cure for cancer, other than its surgical removal, lies in studying how the body defends itself against cancer.

It has been conclusively proved by Ehrlicn and Bashford that no malignant neoplasm ever grows to the extent that its innate proliferative energy entitles it. Hence we find that one of the commonest changes in malignant growth is a retrogressive or necrotic one. Its truly parasitic nature is evidenced by its rapidity of growth and tendency to recurrence in young well-nourished persons, and its decidedly slow growth in the feeble, senile, or diseased. Further, in some persons after operation, or even without it, an inherent force of resistance holds in check the malignant growth.

Since we do not yet fully understand the forces regulating normal tissue growth, much greater is the difficulty concerning abnormal growth. The growth of cancer depends more upon the biological character of the cells than upon their location. The one clinical feature that stands out is the influence of long-continued irritation as an excitant of abnormal growth. So great is this influence that, as far as one can judge, somatic cells without any post-fœtal misplacement or even without any predisposing embryonal mal-development can become transferred into cancer cells under the influence of long-continued irritation. Similarly, experiments with mice showed that animals inoculated with epithelial tumours developed sarcomatous growth in the tissues around the tumour, apparently owing to the irritating influence of the tumour epithelium.

Relative immunisation by inoculation is not yet of any clinical value to us. Further, immunisation inoculation has proved ineffective when tumour growth had commenced, and that is the only condition in which it would serve us clinically.

Anticipatory inoculation, even if warranted by experimental evidence and advocated by us, would not be tolerated by the public.

Educating the public is the first step, for how can we hope to deal with specific instances of disease whilst ignorant of their occurrence. Hedged in with ethics we find it difficult to furnish the public with an intelligent conception of our aims. Similar hesitancy characterised earlier attacks upon tuberculosis. Since enlightenment of the public has been recognised as a first-aid in attack, much good has resulted from co-operative effort.

Similar attack is needed in connection with malignant disease. We lag behind other countries, especially America, where the leading periodical journals have taken up educational campaigns on matters of personal health.

The American Medical Association has assumed this function, and has constituted a Board of Public Instruction for the dissemination of facts bearing on the disease. Yet cautious procedure is essential.

Unlike the tuberculous subject, who is ever hopeful, the cancerous patient becomes stricken with terror when the nature of the disease is known. Our duty will be to enlighten the public concerning symptoms in order to secure their earliest attention. It must not be assumed that every mammary lump is due to cancer, or every atypical menstrual flow due to malignant disease. Such deviations from normal should, however, have immediate attention, and in a very large number of cases assurance from one qualified to express opinion will remove inherent doubts and put patient's mind at ease.

We should make it clear to the public, as it is to ourselves, that notwithstanding all that is being done to investigate the disease, our only hope lies in early and complete removal. We should show them that the growth is parasitic, that it lives on and at the expense of the tissues, that it is a purely local lesion or growth, and that its complete removal, if sufficiently early, will cure the disease.

Moreover, we are warranted in assuring the public, and especially our patients, that the mortality risk from operation in early cases has been considerably reduced by modern surgeons.

Something upon the lines of present attack against tuberculosis should be at once initiated. Lectures before women's clubs, to meetings of nurses, followed by some reference to the lay press within the bounds of ethical procedure, would form excellent adjuncts to Boards of Health and Municipal Councils. Initial prejudices and difficulties regarding tuberculosis have been overcome in a few years. Similar results may be expected in connection with cancer.

The profession as a body may briefly review the position of cancer as applied to women.

One woman in every 8 who reaches her thirty-fifth year dies with cancer; 96 per cent. of all deaths from cancer occur in persons over that age; cancer probably causes more deaths among women than does tuberculosis.

The only known preventive measure is avoidance of long-continued irritation of any kind. When the disease exists, for every victim rescued by early diagnosis and operation, scores pass to death in enduring suffering. Our best surgical results of recoveries in cancer of the breast are about 40 per cent., and of non-recurrence in cancer of the cervix uteri after five years of from 10 per cent. to 20 per cent.

Early diagnosis is essential, and this rests almost entirely with the general practitioner. Three organs should constantly be in his mind as possible seats of malignant disease—the uterus, breast, and stomach.

He should impress upon the women of his clientele the necessity, as they approach the menopause, of reporting to him any changes in the character of their vaginal discharge. If in addition he will make routine vaginal examination at least once a year of all his patients during their child-bearing period, the statistics of operable cancer of the cervix uteri will be brought up to the level of operable cancer of the breast.

It is insufficiently realised that cancer of the cervix does not differ biologically from cancer on the surface of the body, and at an early stage is equally amenable to treatment.

But he will do much more; he will discover many cases of lacerated cervix which undoubtedly predisposes toward malignancy, and should place these in competent hands for repair. The surgeon should in all doubtful cases remove, during operation, sections for examination by a skilful microscopist.

Women should be especially instructed upon the significance of untoward menstrual symptoms, particularly at the climateric epoch.

Germany thus leads by instruction to the public through popular articles in lay periodicals. Winter, in 1891, found that 33 per cent. of cases had been treated for considerable periods of time without examination. A vigorous campaign was then instituted, and in four years the number of unexamined cases found under treatment was reduced by 22 per cent.

In at least 90 per cent. of cases bleeding occurs in some form or other. These patients rarely live more than three years. Three-fourths die within two years, and one-third within one year after the very first reported manifestation. Probably, in Australia, less than 30 per cent. are operable when diagnosed, and of these not more than 15 per cent. are cured by operation for a period of five years.

Cancer in the uterus may develop in the squamous epithelium covering the vaginal cervix, the glands or epithelium of the cervical canal, the glands or epithelium of the endometrium.

Should there occur a bloody or watery vaginal discharge which cannot be definitely accounted for, careful local examination should be made.

When the cervix is rough, friable and bleeding, cancer is usually certain. In all cases a wedge of suspected tissue 1 cm. deep, 3 cm. broad, should be excised and sent to a pathologist. Suppose with such a history one found the cervix healthy, the canal should be carefully dilated, the uterus curetted, and the tissues removed should be at once dropped into a 10 per cent. solution of formalin and sent for examination.

The difference between cancerous and healthy mucosa is so marked, the material is so easily obtained, that no practitioner can exonerate himself from blame who fails to diagnose cancer of the uterus within a week from the first time he sees the patient. The advantage of early diagnosis applies here perhaps more forcibly than elsewhere in the body. Our present results are not good, and our only hope for improvement lies in earlier diagnosis. This will depend upon the zeal of the family doctor and the co-operative aid of women, supported by a well-balanced confidence in microscopy and modern surgery.

Diagnosis of cancer of the breast is based upon careful palpation of the tumour and surrounding breasts, changes in the skin, subcutaneous fat, and nipple. The characteristic hardness and irregularity of outline of the tumour and shortening diameter of the corresponding quadrant of the breast afford almost convincing evidence.

Definite infiltration of the breast about a more or less circumscribed tumour may be almost positively regarded as malignant. Super-added early retraction of a previously elevated nipple is almost pathognomonic.

Atrophy of subcutaneous mammary fat, with some dimpling of the skin over even a small tumour, is significant, and if the whole breast be drawn more closely to the chest wall than the unaffected breast the diagnosis is well established.

I have purposely excluded later definite signs such as adherent skin, ulceration, fungus formation, and glandular enlargement.

Glandular enlargement, *per se*, should never be trusted in the diagnosis of cancer.

Thirty per cent. of these cases are inoperable when first seen, yet every one of them has shown an operable period. We should, with increased enlightenment of the public and co-operation of the profession, reduce without delay this enormous proportion of inoperable cancer of the breast. Every tumour of the breast should immediately be submitted for examination. But the public do not know this, and it is our duty to instruct them. They should be told in the most ethical manner possible that if the tumour is seen sufficiently early, even supposing it to be cancer and the axillary glands be not infected, 85 per cent. will remain well three years after the operation, and probably 75 per cent. be permanently cured; that when metastasis occurs in the axillary glands, and it is sometimes very early, the percentage of cures fall to 30 per cent., and of permanent cures to 24 per cent.; that, later, where metastasis reaches the supra-clavicular glands, the percentage of cures will be further reduced to 10 per cent. and 7 per cent., respectively, or less than this.

Now, 40 per cent. of cases of cancer in women occur in the breast, and we could save a very large proportion did we see them sufficiently early for operative treatment. But, unfortunately we do not.

We endeavour to protect women against tuberculosis, gonorrhœa, syphilis, leprosy, plague, and other diseases, yet we share with the public some innate prejudice and antiquated dread of cancer. We should recognise in this disease an enemy against womankind, should enlighten the public upon its importance, and enlist co-operative help in stamping it out.

Carcinoma of the stomach, like malignant disease of the breast and uterus, is almost always a primary disease distinctly amenable to surgical procedure, and much may be done by the general practitioner not only in its early recognition, but in prevention.

Probably in no organ does operation afford more manifest relief or prospect for cure; yet untreated patients will probably die within one year (8).

Robson found 59 per cent., Moynihan 60 per cent. originating in chronic ulcer of the stomach. Wilson and McCarty, in Mayo's clinic, found by microscopical examination evidence of antecedent chronic ulcer in 71 per cent. There is no doubt that in those cases of relapsing gastric ulcer, early operation will prevent malignant disease. The surest guide to surgery will be the failure of medical treatment to afford relief. In all cases where signs of organic disease is present surgery affords the only hope of cure. But our best hopes lie in earlier diagnosis. For this we need especially the help of the family doctor. We should educate the public to understand that dyspepsia in late adult life must not be regarded as something trivial which has been suffered all patient's life, but something possessing dangerous potentialities towards carcinoma if neglected, and often requiring rest in bed for efficient treatment.

What we must recognise is that positive signs of carcinoma of the stomach do not occur early, and that all of them, except metastasis, may be simulated by other forms of gastric or abdominal disease. My province to-day is chiefly to deal with prevention. I believe, with Sherran, that acute gastric and duodenal ulcer are septic diseases due to oral sepsis, appendicitis, urinary sepsis, burns, or scalds.

Circulating poisons lower resistance of gastric cells or cause minute suppurative foci in lymphatic follicles along the lesser curative. Auto-digestion doubtless follows and leads to ulceration.

Acute gastric ulcer should always be treated by prolonged rest in bed, correction of oral sepsis, careful dieting, light massage for improvement of constitutional tone, and such medical measures as are indicated.

The incidence of cancer of the stomach will thus be markedly reduced. Any ulcer which is large, of long standing, chronic and markedly indurated, or showing the slightest suspicion of malignancy, should be excised.

By thus directing our united attention towards the three organs most liable to cancer in women, by educating the public regarding our desires and aims, and by enlisting confident co-operation, we may hope to reduce the liability to and mortality from cancer in women in a remarkable degree.

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IV.

SECTION OF ANATOMY, PHYSIOLOGY, AND PHARMACOLOGY.

PRESIDENTIAL ADDRESS.

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Syncope, Collapse and Shock.

I HAVE chosen to deal with syncope, collapse, and shock as the subject of my address, not because I can offer to you much that is new, but because it seems to me necessary that someone should make an effort to clear up the indefiniteness which at present appears to surround these conditions. Both in physiological and clinical papers syncope often seems to be termed shock; shock called syncope, and syncope collapse. This is especially confusing to the student. At the same time I wish to avail myself of the opportunity of giving some of the results of my own work in this field of research. I have, however, found that the subject is too large to be dealt with in detail within the scope of an address, consequently I shall be as concise as possible, and shall leave out minor details.

If the picture of profound shock, as seen in cases of railway mangling, be brought to mind, in which reduced sensibility, weakness, pallor, apathy, subnormal temperature, shallow or gasping respiration with duskiness and delayed or absent reflex response to stimulus, all of which combine to portray a condition of general nerve depression, one is not liable to regard shock as being entirely a phenomenon of circulatory depression, as Crile[1] contends, but rather that of general nervous depression, as maintained by Meltzer.[2] Between this state of profound shock and the normal there is a great range of degrees.

The cause of circulatory depression in shock, however, is that which chiefly concerns the clinician, since it is upon a proper understanding of this that successful treatment hangs. The opinion generally held is that it results from vaso-motor paralysis. Crile has done much to emphasise this. Porter and Quinby,[3] however, maintain that the vaso-motor system is not paralysed in shock. They find that for similar nerve trunk stimulation the reflex response bears the same ratio to the blood-pressure in the shocked and in the unshocked animal. Although these statements of ratios are found true by others as well as myself, they by no means necessarily bear the interpretation which Porter and Quinby give them. In the first place, the results may be interpreted as showing that the vaso-motor paralysis is proportional to the blood-pressure; then, again, these ratios hold good only for a certain degree of shock, after which the pressor responses become depressor, or the depressor becomes pressor, and finally cease altogether. Moreover, I find that if the blood-pressure be depressed by bleeding instead of by shock, so that the vaso-motor mechanism is unimpaired, the extent of the reflex rise of blood-pressure is not proportional as Porter and Quinby's contention requires, but it is to the same level in each for similar stimulation. There does not appear to be any justification for the attitude taken by them. Yandell Henderson[4] has recently advanced the claim that acapnia—that is, low CO_2 tension in the blood—is the cause of shock. It is difficult to conceive the possibility of this when we call to mind the partially asphyxial state of the profoundly shocked or the surgically shocked in chloroform anæsthesia wherein the lung ventilation is always minimal and often subminimal; moreover, like Starling and Jerusalem,[5] I have never succeeded in producing appreciable change in the blood-pressure by means of artificial lung ventilation. I have, however, occasioned a fall of blood-pressure in these cases where the pulmonary circulation has been obstructed by excessive artificial respiration, the distention of the alveoli being such as to produce minor degrees of " white lung."

Before proceeding I shall attempt to define syncope, collapse and shock.

1. Syncope is a sudden complete or partial cessation of the circulation from which recovery rapidly occurs, except in cases of advanced myocardial disease, myocardial poisoning, or the profoundly shocked.

2. Collapse is a condition of circulatory depression resulting from sudden loss of blood or of fluid from the blood. If the depression occurs rapidly it may be attended by syncope; further, if the loss of blood be great the heart may cease from low coronary arterial tension.

3. Shock is a state of depression of circulation, respiration, heat-production, &c., which varies from a condition in which a small fall of blood-pressure occurs to one of low blood-pressure with the other indications of profound shock. The onset may be slow or rapid. If recovery occurs, it is relatively slow; if death occurs suddenly, it is from the intercurrence of syncope; if slowly, it is from cardio-vascular failure.

I wish to emphasise that in this classification sudden death in either collapse or shock is always due to syncope as defined above.

From an examination of a fairly large number of experimental records of circulatory changes due to vaso-motor influence, I find that when abnormal afferents reach the central vaso-motor mechanism the rule is that a rise of arterial pressure results, the exception that a fall of pressure occurs.

When the resultant is a reflex rise of arterial pressure, the physiological play consists in the vasor-dilator and the cardio-inhibitory mechanism being thrown out of action, whilst the action of the vaso-constrictor and the cardio-accelerator mechanism is exalted. Consequently, along with the rise of arterial pressure the pulse rate is quickened.

When the resultant is a fall of arterial pressure the physiological play consists in the vaso-dilator and the cardio-inhibitory mechanisms being functionally exalted, whilst the action of the vaso-constrictor and the cardio-accelerator mechanisms is thrown out;

consequently, along with the fall of arterial pressure the pulse is slowed or arrested. This is reflex syncope. The extent of the cardio-inhibitory response largely determines the extent of the fall in arterial pressure.

In deepening shock the cardio-inhibitory mechanism becomes depressed, and then the accelerators come more and more into play. Hence in shock the heart's rhythm increases the lower the arterial pressure, or otherwise the deeper the shock.

This statement, that acceleration of the heart's rate accompanies all sudden reflex rises of arterial pressure, and retardation of rate accompanies all sudden reflex falls of arterial pressure, is at variance with current teaching.

Syncope.

The physiological factors which enter into the causation of reflex syncope have already been stated to be inhibition of the heart and of the vaso-constrictor mechanism. The result of this inhibition represents a fall of arterial pressure, accompanied by slowing or arrest of the heart rate. During this fall in arterial pressure the venous pressure in the large abdominal veins rises. This rise in pressure of the large veins occurs in consequence of an active constriction in the small and medium-sized splanchnic veins, which are promptly emptied into the large ones. These phenomena show that the blood-pressure changes in syncope are not paralytic. In this respect syncope differs very definitely from shock, which, as we shall see later, is essentially paralytic.

The degree of circulatory depression in syncope depends partly upon the intensity of the stimulus, but mostly upon the excitability of the vaso-dilator and vagus mechanisms, especially the latter. This difference in excitability is quite marked in dogs, and clinical experience indicates that it applies also to man. The degree of circulatory depression in syncope varies from a fall of arterial pressure without marked slowing of the heart-rate to one in which the arterial pressure is at zero, and the heart arrested. In the former case the syncope is mostly due to vaso-dilation; in the latter it is mostly cardio-inhibitory. A syncopal fall of blood-pressure when mostly or entirely of vaso-motor origin is denoted by a small, more or less rapid, pulse; when mostly due to vagus inhibition of the heart the pulse is very slow or it is arrested.

Syncope may arise from vaso-constrictor inhibition solely—that is, when the vagus mechanism is depressed, but it does not appear to be possible for it to occur from vagus inhibition solely. When the vagus inhibitory action upon the heart comes into play vaso-dilation occurs simultaneously. This is the more common form of syncope.

Compared with shock, recovery from syncope is rapid and complete. Respiration may fail as a result of cerebral anæmia, but it recovers upon restoration of the circulation.

Proof that syncope is inhibitory and not paralytic is furnished by—(1) Cardiac inhibition ceases upon vagotomy, and is prevented by previous injection of atropine. (2) Vaso-constrictor inhibition ceases promptly upon cessation of the afferent stimulus, and the arterial tension at once begins to return to its former level. (3) The same afferent stimulus which causes syncope at one time may at another time cause a rise of arterial pressure in the same dog and at the same experiment. (4) The peripheral pulse pressure causes a longer oncometric excursion in syncope than in the normal condition, and the excursions increase in length as the arterial pressure falls.

What has been stated refers to reflex syncope, but it differs from other forms only in the source of the stimulus. Emotional stimuli and that resulting from the direct action of poisons such as chloroforms, toxins, &c., and of anæmia of the bulb, produce the same physiological action.

The occurrence of syncope in animals with unimpaired hearts was never found to prove fatal, but when the myocardium had been experimentally damaged by chloroform or other myocardial poison recovery was not constant. Clinical instances of fatal syncope seem to indicate the same factors, more generally from myocardial degeneration.

Collapse.

It is not easily possible to confuse collapse, which is the effect of sudden loss of blood or fluid from the blood, with shock or syncope. The causation is usually so evident, although the physical signs may closely resemble those of shock. The vaso-motor system, at least at first, is unimpaired in collapse, and the fall of blood-pressure entirely due to imperfect filling of the cardio-arteriolar system. If the loss of blood or blood-fluid be sudden and large the syncope of cerebral anæmia usually supervenes. (This, of course, is not that emotional syncope which occurs sometimes at the sight of blood.) Some degree of shock may ensue from defective blood supply to the medulla when the condition is not remedied. It is, however, the heart muscle which fails in untreated cases. Syncope does not fatally arrest the heart in cases of collapse unless there be pre-existing myocardial fault. It appears, from my available evidence, that the pulse is in uncomplicated collapse, unaltered in rate. If complicated by syncope it is slow or it ceases, and if shock follows it is quickened.

Shock.

As already stated, the usual reflex response to afferent stimuli is a rise of blood-pressure. This reflex recurs with each repetition of the stimulus. When the stimulation has been repeated frequently for some time the reflex rise of arterial pressure becomes less and less, until it either ceases altogether or it is replaced by depressor responses. After repeated stimulation it appears that the synaptic resistance rises, so that it blocks afferent impulses more and more until the resistance rises above that of the depressor paths, and then the incidence of the stimulus is on the dilator portion, and reflex syncope occurs. This is evidently the cause of the somewhat rare cases of clinical death by syncope which occur towards the end of a prolonged operation.

In the same manner in the less common instances of primary depressor responses to stimuli, continuance of the stimulus results in cessation of responses or the stimulus passes by the constrictor path, and a rise of arterial pressure results. In these instances, however, the change to pressor responses occurs much more readily than from pressor to depressor. These observations upon the changes in reflex vaso-motor response are confirmed by Hunt.[6]

The circulation is not permanently impaired in either class of response if the stimulation be discontinued at once. But if the stimulation be frequently repeated the responses become less and less, as abovementioned, and the arterial pressure ceases to reach the former level. This form of falling blood-pressure is usually very gradual through a considerable time, after which it becomes more rapid. The period of time during which the arterial pressure is well maintained—that is, before the onset of the relatively rapid fall in pressure—varies greatly in different dogs. It may be less than an hour in one and more than three hours in another, under similar conditions. This appears to apply to man, and is due to variations in the stability of the central vaso-motor mechanism.

That vaso-motor paralysis is largely concerned in bringing about this condition of circulatory depression is indicated by the gradual lessening of vaso-motor response to nerve trunk stimulation: to the stimulus of asphyxia, of cerebral anæmia, of strychnine, of intra-carotid injections of chloroform, &c.

In shock the stored blood and the stored energy of recoil in the large arteries diminish directly with the fall of arterial tension. But although the volume of blood in the large abdominal veins is increasing and the velocity of flow diminishing, the venous tension diminishes almost proportionately with that of the arterial tension. This loss of venous tension plays an important part in the production of shock, since it contributes so largely to the defective filling of the right ventricle. The amplitude of diastolic fall in venous tension varies greatly with the height of the venous pressure. I have found the diastolic fall in venous pressure amount to as much as 22 mm. blood in the inferior vena cava when the venous pressure was equal to 135 mm. blood; and, on the other hand, when the venous pressure was down so as to equal 35 mm. blood the diastolic fall of pressure was imperceptible. The tonus of the veins does not fail quite so early as that of the arteries; the constrictor responses to stimulation are relatively more marked in the veins than in the arteries as shock deepens.

Interpretation of my tracings denotes that the two principal factors concerned in the causation of shock are paralysis of the vaso-motor nervous mechanism which controls the arteriolar adjustment, and paralysis of the nervous mechanism which controls the tonus of the veins.

Among the secondary phenomena of shock are diminution of the excitability and contractibility of heart muscle. This alteration in heart function is the most important of the secondary phenomena. It is what one would expect as a result of the diminished coronary tension. Arrest of the heart is readily accomplished when the arterial tension is low in advanced shock. It even occurs from inhibition produced by adrenalin hydrochloride in ordinary dosage.

The other well-known factors which diminish the rate of diastolic filling of the right ventricle during shock are diminished efficiency of the respiratory pump, loss of tone of the abdominal muscles, and diminished velocity of the venous blood flow.

The progressive form of shock here described is that which is met with in surgical operations—the surgeon's knife, &c., being the source of the stimulus—severe burns, and other rapidly exhausting conditions. Instances in which the stimulus is more violent, such as railway mangling, lightning and electric stroke, &c., show that the arterial and venous tonus are reduced at once to the condition of profound shock, in which the vaso-motor control is practically that of the subsidiary spinal vaso-motor mechanism. The heart in such cases may stop at once from the intercurrence of syncope, brought about by the sudden onset of cerebral anæmia.

The experimental results here given were obtained from animals, and though there are doubtless differences between man and animals in their respective reflex responses to stimulation, it is improbable that there are essential differences in the responses of the three fundamental centres in the medulla.

Treatment of Syncope.

Syncope occurring during operation is almost solely a complication of chloroform anæsthesia either as consequence of overdose or reflexly due to the surgeon's manipulations. In the former case it may be prevented by the exercise of care in the administration, although when the excitability of the vagus and vaso-motor mechanisms is abnormal the margin of safety is narrow. If, in operations upon sensory areas prone to produce depressor reflexes, chloroform must be used as the anæsthetic, the employment of 1-6th to ¼ gr. of morphine, with 1-120th gr. of atropine given hypodermically half an hour before starting the administration will greatly diminish the likelihood of either form of syncope. Reflex syncope may occur in the anæsthesia of ether by the open method, though this is not common. For reasons already mentioned it must be a very rare occurrence in the anæsthesia of ether by the closed method.

Recovery is prompt, the arterial blood-pressure rapidly returning to normal. Operations may then proceed, as the liability to a recurrence of syncope is not great.

When syncope has occurred artificial respiration should be started at once, whether respiration has ceased or not. Artificial respiration releases the heart from inhibition much more readily than would otherwise be the case, besides eliminating some of the chloroform. As venous tension is raised, rotation into the Trendelenberg position is unnecessary as far as the heart is concerned, but it is advisable in order that the medulla be better fed with blood. Heart massage was found experimentally to succeed in restarting the heart in every case of chloroform syncope, but it failed in every case of circulatory failure that was not syncopal—that is, in every case of myocardial paralysis. Intravenous or hypodermic injections are, of course, useless. The horizontal position is usually enough treatment for non-surgical syncope, but occasionally artificial respiration is necessary.

Treatment of Collapse

consists in infusing warm isotonic salt or Ringer's solution slowly and continuously into a vein until the pulse volume is restored. This serves very well in most cases instead of blood infusion or transfusion, and is at all times at hand. Where the loss of blood is extreme the transfusion of blood saves life where isotonic salt solution would fail. The patient should be placed in the exaggerated Trendelenberg position. The infusion of isotonic salt solution is also the treatment of the collapse of profuse diarrhœa.

Treatment of Shock.

Preventive.—The object here chiefly is to block the afferent nerve impuses from reaching the central mechanism. It is only partially effected by general anæsthesia. The various general anæsthetic agents differ greatly in the degree of protection which they afford. The least is attained by the chlorine and bromine substitution products, such as chloroform, methylene dichloride, ethyl chloride, ethyl bromide, &c. Next comes ether by the open method, and then ether by the closed method with or without oxygen. I am unable to confirm Crile's statement that nitrous oxide affords the most effective blockage. I have found it difficult to prevent vaso-motor reflexes from sciatic stimulation by any degree of nitrous oxide anæsthesia short of respiratory paralysis. It is in this respect markedly in contrast with ether. Had Crile said syncope, and not shock, I could readily endorse his statement, since it is so difficult to inhibit the constrictor mechanism when actively stimulated by asphyxia, as happens during the anæsthesia of ether by the closed method and of nitrous oxide, also because the cardio-inhibitory mechanism of the vagus is thrown out of action during vaso-constrictor stimulation, as already stated. As a physiological fact it can be stated that mild asphxia renders ether anæsthesia as by the closed method freer from syncope than anæsthesia by the open method. The pressor effects of asphyxia arise both from CO_2 excess, and from oxygen want in the blood, as Matheson[8] has recently shown. Therefore ether by the closed method with oxygen and nitrous oxide and oxygen produce the same pressor effects as when given without oxygen.

That reflex vaso-motor responses in chloroform anæsthesia are prone to be depressor or syncopal is a statement supported by the authority of Bayliss.[9] When the anæsthesia of chloroform has been in progress the liability to syncope progressively diminishes with the duration of the anæsthesia. It is well, however, to remember that chloroform anæsthesia is never absolutely free from the possibility of depressor reflexes even in the condition of profound shock. The danger varies directly with the severity of the stimulus.

Perineural injections of novocain and injections into parts known to be liable to cause depressor effects during operations, in conjunction with general anæsthesia produce the most effective nerve blocking. This procedure is a valuable surgical auxiliary in both the prevention of shock and reflex syncope. It is advisable to use the minimal quantity of the local anæsthetic agent. For the same reason spinal anæsthesia is a preventative of the surgical shock, whatever its demerits in other directions may be.

Every exhausting factor should be eliminated as far as possible. Pain in cases of operation should be relieved as completely as morphine will admit, as soon as the diagnosis has been made, especially in acute abdominal cases. Fear of operation should be reduced to a minimum by morphine or some soporific. When morphine is to be used prior to the administration of chloroform it should be remembered that morphine depresses the function of the respiratory nervous mechanism. By sufficient dosage of morphine in the case of dogs, this depression is sometimes as marked as that of vagotomy. Morphine, therefore, for such purpose, is contra-indicated in any condition of defective lung ventilation.

During operation the body heat should be carefully conserved. In winter time, especially during prolonged operation, the addition of heat is necessary. Both shock and anæsthesia derange the heat-regulating mechanism. Under experimental conditions the onset of shock is much delayed by keeping the animal immersed in a saline bath at body temperature. Loss of blood should be kept within the lowest possible limits; whilst dragging on, handling or exposing the abdominal organs should not be more than is necessary.

In addition to what has already been said about anæsthetics, it should be remembered that the mode of administration has much to do with diminishing shock. The more perfect the anæsthesia in the earlier part of the operation, and the lighter it is consistent with effectiveness during the after part of the prolonged operation, the less will be the degree of the resulting shock. In operation upon cases already shocked light ether anæsthesia is to be used, and in bad cases with the patient in the Trendelenberg position.

Remedial.—There are four indications:—

(1) Ensure physiological rest by stopping the continuance of the cause.
(2) Maintain the body heat.
(3) Endeavour to secure some constriction of the blood vessels.
(4) Assist the return of venus blood heartward.

(1) Operations should cease before depression has become too great. In non-operative cases pain should be relieved by general and local treatment as in fractures, lacerations, severe burns, &c. Mental rest should be secured in cases of great grief.

(2) Heat added by means of hot water bottles or bags is a familiar method. A better way is that mentioned by Waterhouse. It consists in the use of carbon filament lamps beneath the bed coverings, which are supported on a cradle. This gives uniform warmth to the entire body, and it is very easy to regulate the temperature. It is a common laboratory method of maintaining the body heat of animals during experiment.

(3) We have remedies for raising the arterial pressure in the unshocked, all of which lose this property more or less completely and rapidly in progressively deepening shock. Excepting strychnine, all of them produce their effects by acting locally upon the arterioles. Strychnine, as is well known, produces its pressor effects by stimulating the central nervous mechanism of the vaso-motor system. Crile first showed the inconsistency of using strychnine in shock. He proved that it was worse than useless, because it produced depressor effects in shock if any effect were caused at all. The employment of the other agents is based upon more rational grounds, since they act independently of the central nervous system. Ergot products are useful only in mild shock. The preparations which I have used experimentally have failed completely in moderate and severe shock. The same statements apply to pitui-

tary. The pressor effect of this substance is more lasting than that of ergot, but its depressor quality comes more into evidence as the pressor responses fail. Both pressor and depressor responses to stimuli fail in profound shock. When we come to adrenalin, epinene, and doubtless all of the amines, we find them producing their pressor effects far into the condition of profound shock, but unfortunately the duration of the rise of pressure is brief. This drawback may, to a certain extent, be overcome by slow but continuous infusion into a vein of 1 in 100,000 adrenalin hydrochloride in isotonic salt solution at body temperature, the pulse being used as a guide. Lockhart Mummery[10] has employed the rather indefinite method of leaving some of the solution 1 in 20,000 in the abdominal cavity. Care is necessary, however, in using these pressor substances in profound shock. I have found them arrest the heart permanently in conditions of deep shock, although the quantity administered was not more than a moderate dose for the unshocked dog.

Summarising these results, the employing of strychnine in shock is irrational, whilst that of ergot and pituitary is only indicated in mild and moderately severe cases. They are useless in severe shock. Adrenalin, epinine, and doubtless other amines, are very useful when used as mentioned.

(4) Assisting the venous return: This is best accomplished by raising the trunk considerably above the level of the head, thereby aiding the velocity of venous blood flow by gravitation. In the case of fat patients and those with enfeebled respiration the elevation should be intermittent, and not too long at each period. Oxygen may be needed, especially in such cases. The firm application of an abdominal binder is also of much service in assisting the venous return. Crile's pneumatic suit is of still more service, but it is rarely available. By these means the right ventricle is more readily filled, and the medulla is better supplied with blood. Assisting the venous blood heartward, from the splanchnic bed, seems to me to be the most reliable and efficient measure of treatment, in all forms, but especially in profound shock, in which other remedial measures afford so little help.

The intravenous injection of isotonic salt solution at body temperature is useless in profound shock. A plethoric rise of general blood-pressure may be induced in this way, but it is only temporarily sustained. The fluid rapidly passes out into the tissues, producing general and often pulmonary œdema. This also occurs, but less rapidly, in the lesser degrees of shock.

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SOME CLINICAL CONSIDERATIONS OF OSMOTIC PRESSURE.

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THE cells of all animals above the elasmobranch fishes are bathed in a nutrient fluid whose osmotic pressure is a constant and which is kept constant against alterations in the osmotic pressure of the animal's environment by various mechanisms in the body.(1)

This process of standardisation of the physical constants of the cellular environment has rendered these cells very sensitive to alterations in these constants, and so any alteration in say the osmotic pressure of the environment will be accompanied by alteration in the normal functions or even by disintegration of the cell. For example, the red blood cells of a mammal are attuned to a fluid environment of a constant osmotic pressure which is represented by a depression in the freezing point of about 0·6° C. Now, as it is well known, if these cells be exposed to an environment whose osmotic pressure is above this standard, which is represented by 0·9 per cent. (normal) saline, say to a 5 per cent. salt solution, the corpuscular envelope is unable to withstand the tendency of water to pass out from the cell into the environment in response to its higher osmotic pressure, and so the volume of the corpuscle diminishes and the capsule becomes crenated. Similarly, when the corpuscles are placed in a hypotonic solution or in distilled water, they swell and ultimately burst, due to the passage of water through the capsule into the corpuscle as a result of the higher osmotic pressure of the contents.

The blood of a normal animal has an osmotic pressure represented by a $\triangle$ of about 0·6° C. (which is equivalent to a pressure of about 7 atmospheres). Now all the secretions and excretions of the body have the same osmotic pressure as the blood except the sweat, saliva and urine, and tears, and probably gastric juice. Saliva and sweat are always hypotonic. Urine varies; so also does gastric juice. The tears are said to be hypertonic. The variations in Δ of urine in man lie between 3·5° C. and 0·08° C., the low readings occurring in certain cases of diabetes, or after a prolonged drinking bout, the average on ordinary diet being about 1·2° C. It is principally by means of the kidneys, with the intelligent co-operation of the will, that the osmotic pressure of the blood is maintained constant. Any tendency to dilution of the blood is quickly and easily got over by the copious production of watery sweat and dilute urine, but the reverse process, the keeping down of the osmotic pressure when circumstances determine a rise is much harder, and entails a great deal of work on the kidneys in producing a concentrated fluid from the blood against a great osmotic pressure. That the cells of the body soon suffer when the osmotic pressure of the blood rises is at once seen in the case of sailors lost at sea who try to quench their thirst with sea water, which has an osmotic pressure of about four times that of the blood. The cells of the highest nervous centres are the first to suffer, and the individual becomes maniacal, and usually puts an end to his existence long before his suffering but wiser comrades who do not drink the sea water, but these eventually suffer the same fate, especially if atmospheric or other conditions favour the loss of abnormal quantities of water in the form of perspiration, &c. Similarly with other tissues—*e.g.*, if a strong saline or saccharine solution be taken into the mouth the mucous membrane becomes "roughened," or if any part of the body be kept submerged in water, water is imbibed and the part becomes water-logged.

Now the passage of salts or water through a tissue in response to osmotic influences acting from without is accompanied by more or less painful sensation. It is generally common experience that the introduction of a drop of distilled water or strong salt solution into the conjunctival sac—especially if this membrane be inflamed—is distinctly unpleasant, while the introduction of normal saline, especially if it be warmed to body temperature, is quite unnoticeable. This is a point which could very well be noted in connection with the ordering of eye lotions or eye washes, a drug having a much better chance of exerting its action when the eye structures exposed are not being irritated by a hypotonic solution. And similarly with solutions whose only functions are lavage, such as lotions for washing out bladder or urethra, or for bathing extensive raw surfaces. I have personally tried isotonic solutions in all these classes of cases, and the patients' expressions of comfort were really worth the trouble of adding a teaspoonful of salt to the pint of lotion. Another use to which a normal saline solution could be put is in the making up of washes and lotions in which the surgeon steeps his hands for several minutes at a time in sterilising them. As is well known, under ordinary conditions, the skin of the hands soon becomes water-logged, and loses the delicacy of its sense of touch which is a distinct disadvantage in some cases.

We thus see how the various tissues of our body suffer when exposed to hypertonic and hyptonic solutions. The walls of the mucosa lining the alimentary tract form no exception to this rule. Although the semi-solid and solid foods which enter our alimentary canal are principally albuminous, and hence play a very small part in the osmotic phenomena in the body, yet we know that liquid foods which enter the body have an osmotic pressure varying from practically nil in the case of tap water, to many times that of the blood in the case of some beverages. What is nature's attitude towards the ingestion of these foodstuffs? My researches on the subject (2) have shown that of all the fluid foods which enter the body, the sense of taste regards as bland and acceptable all whose osmotic pressure is a little below that of the blood (with a $\triangle$ of about 0·4° C.–0·5° C.), so long as the fluid does not contain any irritating chemical substances which have specific action on the senses of taste or smell.

Fluid foods which possess a lower osmotic pressure than this, such as extracts or infusions of vegetable or animal matter, such as tea, coffee, vegetable soups and meat broths, are brought up to this standard by a sugar or salt addition, respectively, in the various cases. This hypotonic food leaves a water excess which is useful to the animal's economy for the dilution of hypertonic gastric juice or the supplying of a means of flushing out the system through the secretory activity of the cells of the kidney or sweat glands. The ingestion of foods whose osmotic pressure is above that of the blood leads to the development of the sense of thirst. This desire for drink is most naturally satisfied by water which would dilute the hypertonic solution down to a reasonable degree. As far as my work has gone, it shows that alcoholic beverages form the only exception to this rule. The explanation is that man is the only animal who manufactures and consumes alcoholic beverages, and so nature has not had time to develop in him the protective mechanism in this *osmotactic* quality of the sense of taste which demands the ingestion of water to satisfy the thirst brought on by the taking of the hypertonic food into the mouth. All fluid foods which contain a saline content above that of the blood must enter the body under protest. In the case of saccharine fruit juices or alcoholic beverages, the sugar or alcohol—as the case may be—rapidly enters the blood and is oxidised or stored in the body while the saline constituents alone remain to play their part in the osmotic phenomena in the body. My work has shown that all those fruit juices which possess a high osmotic pressure, owe this high pressure to their sugar and organic acid content and not to their inorganic salts.

These facts lead us to the conclusions that in the ordering of diets, particularly for infants or invalids, especially those afflicted with gastro-intestinal troubles, we must see that they are correct not only as to their chemical constitution but also as regards their physical relationships to the blood. The broths, drinks, &c., should always be salted or sugared up until they are just hypotonic to blood—thus allowing a water excess for flushing out purposes. The foods should not even be isotonic with the blood, for the experiments of Sommerfeld and Röder (5) have shown that an infant fed on an isotonic milk diet excretes hypertonic urine. This means that under these *apparently* ideal conditions the kidney is doing work against osmotic pressure. This is a consideration in cases of severe kidney inefficiency for, by so modifying the diet, we can diminish the work of this organ against osmotic pressure and so give it a better chance of recovering and performing its other functions more effectively, for the work of Kövesi and Roth-Schulz (3) shows that the pathological kidney does not possess the power of accommodation (Akkomodations breite) that the normal kidney possesses, particularly those described as acute and chronic parenchymatous nephritis.

In diarrhœic conditions, too, the osmotic pressure of the diet should be taken into account. The transudate in cholera and diarrhœic conditions generally has been shown to be really a saline fluid isotonic with blood, so that the fluid which is given to the patient to quench the severe thirst produced should not be pure water, but should be a fluid containing all the saline constituents of the blood. Such a fluid as Ringer's Fluid (4) slightly diluted, would admirably suit this purpose.

The whole subject is intensely interesting and opens up many fields which can, however, be much better studied clinically than in th laboratory.

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4. Ringer's Fluid.—

NaCl	9·00 gm.
KCl	·42 gm.
$CaCl_2$	·25 gm.
Na_3CO_2	·10 gm
Water	1 litre

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THE PERMEABILITY OF LIVING MEMBRANES AND THE DIFFICULTIES OF A PURELY PHYSICAL THEORY OF PERMEABILITY.

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The Difficulties of a Purely Physical Theory of the Permeability of Living Membranes.

The early work of Graham, who showed that the velocity of diffusion of a gas is inversely proportioned to the square root of its density, and who divided soluble bodies up into two classes—the crystalloids, that would pass through an animal membrane, and the colloids that would not, was followed by the pioneer work of Pfeffer on osmotic pressure, while the remarkable parallelism between the properties of gases and those of dissolved substances was emphasised by van'lt Hoff in 1887. The plasmolytic method of making isotonic solutions of different substances was first employed by De Vries in 1884; he used cells taken from the epidermis of *Trasdescantia discolor*. Much has been done since the early work of these investigators to find replies to such problems as: wherein lies the efficiency of a semipermeable membrane? why is a membrane permeable to one substance, impermeable to another? and to answer such qustions as have a direct bearing, not only on the purely physical side of osmotic pressure, but also on the osmosis which plays such an important part in the equilibrium between plant and animal cells and their surroundings.

It has to be recognised, however, that in the present state of our knowledge, the behaviour of the living cell membrane towards the substances with which it comes in contact is, in many cases, incapable of interpretation on a purely physical basis. The following examples may be mentioned:—

1. The permeability of a cell membrane alters on the death of the cell; certain dyes can enter the cell only when the latter is killed.
2. The inorganic constituents of the blood corpuscles are notably different from those of the plasma in which they float, and from which their constituents must be derived. The corpuscles are comparatively rich in potassium and phosphate, while the plasma is poor in these, but rich in sodium and chloride.
3. Tadpoles placed in 0·6 per cent. solution of sodium chloride remain normal. If transferred to a 0·8 per cent. solution of sodium chloride they lose water and shrink considerably in size in twenty-four hours. If now transferred to a solution hypotonic to a 0·8 per cent. solution we should expect them to increase in size; however, they do not; it therefore appears that the epithelial membranes of tadpoles are permeable to water in one direction, but not in another.

4. In the processes of secretion, owing to the special activity of the secretory organs, a substance may be transferred from a place where its concentration is low to a place where its concentration is high. For instance, in the kidneys urea is transferred from the blood, where its amount is small, to the urine, in which the proportion of urea is much larger.
5. In plants whose roots are in contact with the very weak solution in water of the soluble constituents of the soil, these constituents are daily transferred to the more concentrated protoplasm within the roots.

In such cases not only is the equilibrium, which experience based on experiments with non-living material would lead one to expect, never attained, but the transference of material is in the reverse direction to that which, on purely physical grounds, one would prognosticate. The limiting membranes of the living cells do not behave like the dead parchment paper or pig's bladder used for experimental purposes, but in a manner quite different, and I therefore propose to discuss in some detail the relation of the cell to its limiting membrane.

According to Traube[1], who discovered and studied various precipitation membranes, such as copper ferrocyanide and gelatin-tannin, the feature of a semipermeable membrane which enables it to differentiate between one substance and another is the size of its molecular interstices. Acting like a sieve, the membrane prevents the passage of particles which have a relatively large volume. Compounds which have figured in direct determinations are mainly carbohydrates and ferrocyanides, and if one assumes that the volume of a molecule depends on its weight and complexity, their behaviour lends support to Traube's conception.

There are, however, many facts which are opposed to the sieve theory of the membrane.

In an investigation of the permeability of membranes composed of gelatin-tannin, zinc ferrocyanide, and copper ferrocyanide, Tammann[2] found that of seventeen dyes tested eleven penetrated the first membrane, seven the second, and five the third. On the basis of the sieve theory, this would mean that the interstices or pores were widest in the gelatin-tannin membrane, and narrowest in the copper ferrocyanide membrane; but with individual dyes it was found that in some cases the copper ferrocyanide was more permeable than the zinc ferrocyanide; in other cases the latter was more permeable than the gelatin-tannin membrane, a result quite inconsistent with the sieve theory.

Again, Raoult[3] found that when methyl alcohol and ether are separated by a membrane consisting of pig's bladder, there is an osmotic flow from the alcohol to the ether. If, however, the two liquids are separated by a membrane of vulcanised caoutchouc, osmosis takes place in the opposite direction—that is, from the ether to the alcohol. There must, therefore, be some other factor involved besides the pores of the membrane. The nature of this factor is clearly indicated by Tammann's[1] experiments, which showed that pig's bladder absorbs ten times as much methyl alcohol as ether, and that caoutchouc absorbs about 100 times as much ether as methyl alcohol. The direction of the osmotic flow is therefore determined by the preferential absorption of one of the two liquids by the membrane.

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[1] Archiv. Anat. Physiol., 1867, 87.

[2] Zeit. Physikal. Chem., 1892, 10, 255.

[3] Zeit. Physikal, Chem., 1892, 10, 255.

The result was established more definitely by Flusin[2], who measured the velocity with which water, methyl alcohol, and amyl alcohol pass through pig's bladder when the other side is bathed with ethyl alcohol, to which the bladder is practically impermeable.

It is possible to construct osmotic cells in which absorption by the membrane is undoubtedly the ruling factor.

The efficiency of a semipermeable membrane depends apparently on its ability to differentiate, by solvent or absorptive power, between the substances which seek to penetrate it. When we inquire how far this view can interpret the phenomena connected with the permeability and impermeability of living membranes the conditions met with are infinitely more complex.

In this field Overton's work[3] stands out pre-eminently. He worked mainly with plant cells, but there is great agreement between plant and animal cells in this respect that a substance which can penetrate the one can also penetrate the other.

Using the plasmolytic method upon plant cells, he found that all monohydric alcohols, aldehydes, ketones, and esters have the power of rapid penetration.

The dihydric alcohols and the amides of monobasic acids penetrate the cell membrane more slowly, glycerine and urea still more slowly.

Hexahydric alcohols, the hexoses, the amino-acids, and the neutral fats of the organic acids can hardly penetrate at all.

Using the tannin naturally contained by many plant cells as an indicator, inasmuch as it forms precipitates with aqueous solutions of many chemical compounds, Overton found that ammonia, aliphatic amines, and free alkaloids penetrate the membrane with ease, while for the salts of the alkaloids the membrane is less permeable. Basic aniline dyes are in general easily taken up by animal and plant cells, whereas those which are sulphonic acid salts either cannot penetrate the membrane or do so with great difficulty.

Overton concluded that so far as the living membrane can be regarded from the purely physical standpoint it is selective absorption on the part of the membrane which determines the ability or inability of any substance to enter the cell.

Overton maintains that the membrane is impregnated with cholesterol, or a mixture of this substance with other compounds such as lecithin, and that the ability of a substance to make its way into the cell depends upon its solubility in cholesterol. There is a distinct parallelism between the rapidity with which various substances penetrate the cell and the extent of their solubility in cholesterol and lecithin solutions. The theory does not account for the fact that—

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[1] Zeit. Physikal. Chem., 1897, 22, 490.

[2] Compt. rend. 1898, 126, 1497 ; 1900, 131, 1308.

[3] Vierteljahrschrift Zurich, 1895, 40, 99 ; 1899, 44, 88 ; Zeit. Physikal. Chem., 1897, 21, 189 ; Jahrb. wiss. Botanik. 1899, 34, 669.

1. Cell membranes are readily permeable by water.
2. Simple inorganic salts often cannot penetrate the membrane, even though it be impregnated by water.
3. There are dyes insoluble in cholesterol which can penetrate the membrane.

When we consider the nature of the substances which come in contact with the cell wall we find they may be classified, briefly, as electrolytes, colloids, and enzymes.

Electrolytes.—According to the modern theory of solution, an electrolyte when dissolved undergoes a certain amount of ionisation, the degree of ionisation being greatest in comparatively dilute solution.

For present purposes we may define *ionisation* as a term connoting the separation of a substance in solution into several portions, each capable of acting as a distinct kinetic unit, those units with a positive electrical charge being known as cations, those with a negative charge as anions; a *catalyst* as a body which alters the rate of reactions; and *enzyme* as a catalyst of colloidal nature produced in living organisms.

The fluids which bathe the frames of plants and animals are electrolytic solutions.

Under the influence of enzymes changes take place in the organism which result in the production of ionised from non-ionised substances.

Proteins, for example, are split up by the action of trypsin, and produce peptones and amino-acids, substances which are ionised to a certain extent.

It appears that separation by diffusion of the ions of an electrolyte, on account of the greater mobility of the one than the other, cannot take place to any great degree. With hydrochloric acid, for example, in consequence of the greater mobility of the hydrogen ions, the front rank of the diffusing acid will consist of positive hydrogen ions, while behind them will be an excess of negative ions. Electrostatic forces are thus called into action which prevent anything more than an infinitesimal separation, and which have the effect of retarding the advance of the hydrogen ions and accelerating that of the chlorine ions.

The difference in mobility of various ions is modified, so far as diffusion is concerned, by the electrostatic attraction between the ions, and gives rise to a difference of potential at the common surface—(1) of salt solution and water; (2) of differently concentrated solutions of the same salt; or (3) of solutions of different salts.

In this direction must we seek the explanation of the electrical effects which, as found by physiologists, so frequently accompany vital activity. Differences of electrical potential in the tissues are probably due to a separation, infinitesimal in extent, of the positive and negative ions of the electrolytes which bathe the tissues.

It may be possible that the two ions of a salt are characterised by a difference in power of penetrating the living membrane. If a membrane were bathed by water on one side and by a salt solution on the other, then, if the anion could pass the membrane, but not the cation, only an infinitesimal separation would be brought into play, although the membrane would be the seat of a difference in potential which may have a bearing on the problems of electrophysiology. In the case of the salt just described the anions are prevented from passing through the membrane by the inability of their positive partners. Actual transport of these anions through the membrane would be rendered possible either—

1. By adding to the salt solution another electrolyte, the cation of which is able to penetrate the membrane; or
2. By adding, to the water on the further side, a salt, for the anion of which the membrane is permeable.

In the first case the cation of the added salt and the anion of the original salt could cross the membrane together in electrically equivalent quantities; in the second case there would be an exchange of the two anions, also in electrically equivalent quantities.

Such a picture is not wholly imaginary, for investigations by Hamburgh and Koppe have shown that the protoplasmic membrane of blood corpuscles is generally permeable to anions. If blood corpuscles are separated by centrifuging, suspended in an isotonic solution of sodium chloride, and subjected to a current of carbon dioxide, the salt solution becomes strongly alkaline. The most satisfactory explanation of this phenomenon is based on the view that the carbon dioxide penetrates the covering of the blood corpuscles, and, reacting with some of the contents of the corpuscles, probably the proteins, gives rise to the carbonate ions $H\ CO_3$ and CO_3. The plasmatic membrane being permeable to anions, an exchange between the carbonate and chlorine ions in the surrounding fluid becomes possible, and leads to the production of sodium carbonate, and consequent alkalinity in the previously neutral sodium chloride solution.

In the plant world a somewhat similar phenomenon is manifested.

Hall,[1] referring to the use of nitrate of soda as a manure on the experimental plots at Rothamsted, says: "One of the most characteristic effects of the use of nitrate of soda as a manure, either repeatedly or in any quantity, is its deleterious action upon the texture of a heavy soil, which loses its tilth and cakes badly. The explanation appears to be that when a plant is feeding upon a neutral salt like nitrate of soda, it takes up more of the nitric acid than of the soda, leaving behind in the soil some of the soda combined with carbonic acid excreted from the root. Water cultures in which plants are grown with nitrate of soda will actually become alkaline to test-paper from this cause."

When ammonium salts are used as a source of nitrogen, the amount of calcium carbonate in the soil is reduced. At the farm of the Royal Agricultural Society, at Woburn, where, through the continued use of ammonium salts as manure, the soil is actually acid to litmus paper, and refuses to grow barley any longer, the former fertility is at once restored by an application of lime. The acidity of the soil where the ammonium salts have been used is due to the attack of various moulds and other micro-fungi upon the ammonium salts; they seize upon the nitrogen for their own nutrition and set free the acids with which the ammonia was combined; if there is no calcium carbonate present to neutralise the acids they combine with the calcium of the humus and set free humic acid, which accumulates from year to year.

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[1] A. D Hall, Fertilisers and Manures, 1909. p. 54.

Some Properties of Colloidal Solutions.

If now we examine the behaviour of colloids we find that they, too, are much influenced by electrical phenomena.

In dealing with any colloïd it must be borne in mind that its behaviour depends upon its previous history. In support of this contention I may cite the three following examples:—

(**a**) If a gelatine solution, after being kept for a short time at 70-80 degrees, is cooled, say, to 25 degrees, the value then observed for its osmotic pressure is considerably higher than it was before the solution was heated. Only after the solution has been kept some days is there a return to its former value. The osmotic pressure exhibited by a gelatine solution is, therefore, to some extent dependent on its previous history.

(**b**) A colloid belonging to the suspensoid class gives, with water, a mixture which is non-viscous and non-gelatinising, but is coagulated on the addition of small quantities of electrolytes. A colloid belonging to the emulsoïd class gives, with water, a mixture which is viscous, gelatinises, and is not readily coagulated by salts.

With a colloid belonging to the former class an amount of salt which is not capable of causing immediate coagulation is, nevertheless, effective after a certain interval, and the total quantity of electrolyte required to bring about complete coagulation of the suspensoid varies according as the electrolyte is added all at once or in several portions successively.

(**c**) *The Danysz Phenomenon.*—The toxicity of a mixture of diphtheria toxin and antitoxin depends on the way in which the two are mixed. If the amount of toxin added is such that the mixture is untoxic, then in a second experiment, in which the same amounts of toxin and antitoxin are taken, in which, however, the toxin is added in instalments, the resulting mixture is toxic. This phenomenon is exactly analogous to what happens in the precipitation of a colloid by an electrolyte, or in the precipitation of one colloid by another; the amount of electrolyte or colloïd required for complete precipitation varies according as it is added all at once or in instalments. The condition of a toxin in antitoxin mixture, therefore, resembles that of colloïdal solutions in that it is not completely defined by a statement of its composition; *its character depends on its previous history.*

The great majority of colloïdal solutions, when examined in the ultra-microscope, are found to contain distinct particles.

As a rule, colloïds carry a definite electrical charge.

The question indeed suggests itself: Is not the Brownian movement due to the particles having each a similar electric charge?

Protein in an electric field may be either electro-positive or electro-negative.

Egg albumen in an alkaline medium was found to be electro-negative, but in an acid medium electro-positive. If the coagulum formed at the anode by serving a current through the original alkaline fluid is thoroughly broken up the particles leave the anode and move towards the cathode; their electrical character has changed.

It was Hardy[1] who first realised the full significance of the effect of an electrolyte upon a colloid, and formulated the rule that the ion of an electrolyte which determines the coagulation of a colloidal solution is the one which has a charge upon it opposite in sign to that on the colloidal particles.

Since the coagulation of a colloid by an electrolyte is due to the neutralisation of the electric charge upon it, a positive colloid added to a negative colloid should produce coagulation; this is found to be the case, and the precipitation of egg albumen by solutions of various complex acids, *e.g.*, molybdic, tungstic, and tannic acids is of this nature. All the evidence goes to show that the precipitation of a suspensoid by an electrolyte is essentially electrical in character; the precipitation of a reversible colloid by the neutral salt of an alkali metal, on the other hand, has much in common with the phenomenon of "salting out," as practised by the organic chemist. Each ion is found to be responsible for part of the effect which would not be the case if the phenomena were electrical.

Solutions of reversible colloids approximate more to true solutions than do solutions of suspensoids.

When a general solution of protein is made slightly acid or slightly alkaline its properties undergo a marked modification. In a potential gradient the protein now migrates towards the anode or cathode, according as the medium is alkaline or acid, and in respect also to the precipitating powers of salts its behaviour now resembles that of an ordinary suspensoid.[2]

Small quantities of acids or alkali are unable to precipitate carefully dialysed neutral protein, but as soon as the protein has acquired an electro-negative character it is readily precipitated; the precipitation, further, is irreversible in character, and therefore quite different from the precipitation of neutral proteins by alkali salts.

The readiness of protein to change its character with the acid or alkaline reaction of the medium becomes intelligible on the basis of Hofmeister's theory that the proteins are produced by the condensation of several amino-acids, and that the protein molecule is characterised by the presence of at least one amino group and one carboxyl group. On this view, protein is an "amphoteric" electrolyte—that is, an electrolyte which may act either as an acid or as a base, which may split off hydrogen or hydroxyl ions.

According to circumstances, therefore, the protein molecule may assume either an acid or a basic function; it forms salts both with bases and with acids.

When a reversible colloid is added to the solution of a suspensoid, the precipitation of the latter by electrolytes is more or less inhibited —*i.e* their action is protective.

Enzymes, toxins, and antitoxins are all colloids.

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[1] Zeit. Physikal. Chem., 1900, 33, 385.

[2] Hardy, Zeit. Physikal. Chem., 1900, 33, 385.

Two factors enter into any interpretation of the relation between a colloid and its medium—firstly, electrical phenomena, which have already been discussed; and secondly, the relatively enormous surface of contact between the colloid and its environment. We may regard the interaction of colloids with each other and with various solid and dissolved substances as being essentially a process of *adsorption*, this term being used to describe the concentration at the *surface* of a colloid of a substance introduced into a colloidal suspension; and it is in regard to this local concentration at the surface (which in colloids is enormously developed) that adsorption differs from absorption.

The opinion has rapidly gained ground that where mixtures of colloids and ions are involved, as in the living cell, the equilibrium between these particles is largely of the nature of an absorption equilibrium. In such a complex case it is, of course, impossible to say exactly what part is played by the chemical and physical factors respectively, but the study of proteins is showing that these substances, which are so essentially associated with the living cell, are peculiarly liable to exhibit absorption phenomena. Not only are proteins readily absorbed by charcoal, mastic suspension, kaolin suspension, &c., but they themselves appear to absorb electrolytes from solution.

For many of the references and examples in the foregoing I am indebted to the work of J. C. Philip on "Physical Chemistry."

Some Properties of Enzymes considered as Colloids.

Of the various hypotheses put forward to explain catalysis one is of importance in connection with colloidal catalysts such as enzymes are, namely, surface condensation (adsorption) of the reaching bodies, in which case the accelerated rate of change is, in all probability, due to increase of concentration. A similar hypothesis was suggested by Faraday to explain the action of platinum on mixtures of oxygen and hydrogen gas. In many cases an enzyme acts as an accelerator of a change which, perhaps, given sufficient time, would take place without it; for example, Brailsford Robertson noticed that solutions of ammonium caseinogenate slowly increased in electrical conductivity when left to themselves, a change similar to that which occurs when they are acted upon by trypsin; and starch solutions undergo spontaneous change in the direction of dextrin and sugar.

Statements have been made to the effect that enzymes are electrically charged. Probable as this is, it is perhaps premature to accept it until we know that we have pure enzymes in our hands.

Enzymes are colloids, and have the property of carrying down with them constituents of the solutions from which they are precipitated. It is therefore not to be wondered at that amylase or invertase as obtained in the usual way give carbohydrate reactions and that pepsin or trypsin give protein reactions. The more they are purified the fewer characteristic actions do they show, and the more unstable do they become.

In the purification of enzymes by dialysis it sometimes happens that the solution loses its activity. Diffusion through the parchment paper, destruction of enzyme, or removal of co-enzyme may be the cause.

It has been shown by Bierry, Giaga, and V. Henri[1] that if pancreatic juice be dialysed it loses its power of acting upon starch or maltose. The addition of certain electrolytes restores this activity. By testing various salts it was shown that the electro-negative ion is the only potent one, and among these ions the chlorine or bromine ion is the essential one. Thus sodium and potassium chlorides are active, while the sulphates are inactive.

Pavy and Bywaters[2] have recently shown that the inverting power of yeast is increased tenfold by addition of boiled extract of yeast. This activating effect is shown to be attributable to the acidity of the boiled extract, equivalent results being obtained by the substitution of acetic acid for the boiled extract.

The diastatic enzymatic agent in the oyster was found to be influenced in the same manner.

As with invertase, basic substances were found to exert an inhibitory influence over diastase, and the inhibitory effect could be removed by additions of acid.

A considerable number of enzyme actions are reversible. There is no doubt that lipase action is reversible. The observations as to protein synthesis in the organism distinctly indicate a reversible enzyme action.

Bearing in mind these facts, viz., the adsorption phenomena exhibited by enzymes, the influence of electrolytes upon them, and the reversibility of their reactions, the difficulty of interpreting on a purely physical basis the passage of a substance into or out of a cell through its limiting membrane becomes apparent.

In living cells only comparatively few are dry on one side; in other cases the cells are either covered with a slime or their protoplasm is continuous.

The cells, for example, of the intestine, the root hairs of plants, the gills of fish, and the cells of most bacteria, are covered with a slime. The bacterial slime appears to be the effective agent in clarifying water passed through filter-beds of sand, the surface of the sand becoming covered with a more or less gelatinous skin.

" The researches of Carnoy and other cytologists have demonstrated that the constituent cells of multicellular organisms are knit together by protoplasmic cell-bridges (ponts intercellular), in which cell membrane, protoplasm, and sometimes even nuclear filaments are involved (Ide). Hence it may be inferred that besides serving as nutritive channels, these bridges also transmit physiological impulses from cell to cell."[3]

It is a comparatively easy matter to demonstrate the continuity of the protoplasm from cell to cell in the thick-walled date stone, and in other vegetable structures.

From the first of the above-mentioned phenomena, the presence of a slime outside the cell wall, to the actual protrusion of protoplasm in the form of cilia through the cell wall as in bacteria, or as cell bridges in multicellular organisms, there appears to be every gradation, and hence to the slime, consisting of electrolytes, colloids, and enzymes, practically in continuity with the protoplasm, I would attribute primary importance, and to the cell wall, on which it lies, secondary importance, as being a mere skeletal supporting structure which, *per se*, has little influence on what passes through it in one direction or the other.

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[1] Compt. rend. Soc. de Biol., 1906, 60, 479.
[2] Journal of Physiology, Vol. XLI, Nos. 3 and 4.
[3] The Natural History of Cancer. W. Roger Williams, 1908, p. 206.

Most disinfectants show different Rideal-Walker coefficients when tested against different organisms. For example, a well-known disinfectant has the following carbolic acid coefficients for the organisms specified:—

B. typhosus	11·0
S. pyogenes aureus	9·3
B. pestis	34·0
B. tuberculosis	11·0
B. dysenteriæ	10·0
B. diptheriæ	20·0
Vibrio choleræ	32·3
B. mallei	15·0[1]

This difference can scarcely be accounted for on the modern method of reckoning, that a definite amount of toxin is required to cause the death of a definite body weight, and although the bacteria mentioned are not capsulated, the reason of the difference in the coefficients, which vary from 9·3 to 32·3, is probably to be sought in the protective covering rather than in the thickness of the cell wall.

Steven Bierema,[2] experimenting with soil bacteria to discover the efficacy of various culture media, found that, while sodium nitrate and ammonium nitrate in the presence of certain carbohydrates are readily assimilated, magnesium ammonium phosphate proved to be one of the best sources of nitrogen, notwithstanding its very slight solubility.

Again, from the constituents used for making water cultures of plants, ammonium magnesium phosphate is frequently precipitated as the result of double decomposition; nevertheless, it appears to be a source of nutriment.

That a practically insoluble salt like ammonium magnesium phosphate can be used as a source of nitrogenous food both for bacteria and for plants appears to me to show that the slime, external to the living cells, consisting of electrolytes, colloids, and enzymes, has functions more important than are usually attributed to it.

There is reason to believe that in the process of digestion, during which the stomach does not digest itself, mucin plays a somewhat prominent part.

Weinland[3] stated that antipepsin exists in the gastric mucous membrane, and antitrypsin in that of the intestine. These bodies are supposed to confer upon these tissues their immunity from attack by the digestive juices. Hamil and Bayliss could not confirm the presence of antitrypsin in the intestinal mucous membrane, but Hamil confirmed the presence of antipepsin in the gastric mucous membrane.

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[1] W. Partridge, The Bacteriological Examination of Disinfectants, 1907, p. 34.
[2] Centr. Bakt. Par., 1909, ii, 23, 672–726.
[3] Zeit. f. Biol., 1903, 44, 45–60.

According to Klug,[1] the active body which has the power of protecting the cells of the mucous membrane is the mucin, which is always present in considerable quantity. This it does by forming adsorption compounds with the enzymes.

Solution of Substances in the Absence of a Membrane.

I have shown previously that even an insoluble salt like ammonium magnesium sulphate can be taken up by cells enclosed in a membrane.

When one comes to the examination of those substances that can be taken up by protoplasm destitute of a membrane, the work of Loeb on the artificial fertilisation of the eggs of the sea-urchin attracts the attention.

When a spermatozoon enters an egg cell of a sea-urchin, it seems to cleave a sort of passage through the egg, leaving a distinct trail behind. Almost the instant that it enters the egg a peculiar appearance is observed. The egg itself is rather granular and opaque, but immediately after fertilisation a small clear area begins to appear on the outside, which gradually increases in width until there appears a granular sphere enveloped in a clear, transparent shell. This clear area is surrounded by a membrane which prevents any more spermatozoa from entering, so that, as a general rule, and under normal conditions, only one spermatozoon enters the egg.

Professor Loeb first fertilised artificially the eggs of the sea-urchin on the Atlantic Coast; with the sea-water of the Pacific he failed. Failures were also recorded at Plymouth, in England, but when a little alkali was added, in each of these cases successful artificial fertilisation was effected, but *clear* membrane was not produced. If, however, the eggs were first treated with carbonic acid, formic acid, acetic acid, propionic acid, or butyric acid, then a clear membrane was produced.

Process.—Two c.cm. of a very weak solution of butyric acid are added to 50 c.cm. of sea-water, and in this mixture the unfertilised eggs are placed for two minutes. The eggs are then placed in concentrated sea-water, 60 per cent., for fifty minutes at the ordinary temperature; they are then placed in normal sea-water. Directly after they have been taken out of the butyric acid solution they form a clear membrane. When they reach the ordinary sea-water they develop normally. Delage, of Paris, raised adult sea-urchins from artificially fertilised eggs.

From the present outlook two important points emerge:—

1. All the evidence goes to show that with all the various acids tried it is the undissociated molecule which penetrates the egg and brings about the formation of the membrane.
2. That treatment with an acid must be followed by treatment with an alkali, and knowing, as we do, that alkali increases the velocity with which oxidations occur in animal fluids, there is little doubt that the function of the alkali in fertilisation is the acceleration of the oxidations necessary for cell development. When the egg cell is undergoing oxidation there is very little doubt but that the lecithin it contains is split up, and that the cholin is set free.

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[1] Arch. Internat. de Physiol., 1907, 5,207-217.

There is reason to suppose that when a cell nucleus divides into two there is a concentration of cholin between the two daughter nuclei.

While the cell wall acts as a skeletal structure, strands of protoplasm pass from cell to cell. If a cell wall be damaged by excision the part excised is frequently regenerated. In the higher animals this is only true to a limited degree; in the lower animals, especially among those that live in damp situations, crustacea and amphibians, whole limbs may be regenerated, the aggregated forces of the body being supposed to control the formative processes going on in each part. Particular tissues must be present; if a newt's leg be cut sharply across it will be regenerated, but if removed by severing the muscles around the scapula it will not be regenerated.

If the cell membrane be continually destroyed, as in the case of a gnawing larva in plant tissues, where doubtless the protoplasm is impregnated at the same time with the salivary secretion, excessive cell multiplication may take place resulting in the formation of galls.

All the larvæ which I have examined have an extraordinary amount of this secretion, which, when irritated, they will force out from their mandibles as a large, usually highly coloured, drop.

The galls produced by the same insect in the alternating phases of its heterogenesis are markedly different, and hence the stimulus appears to be some specific secretion of the larvæ themselves.

Darwin, in discussing the subject, remarks:—"As the poisonous secretion of insects belonging to various orders has the power of affecting the growth of various plants; as a slight difference in the nature of the poison suffices to produce widely different results; and lastly, as we know that the chemical compounds secreted by plants are eminently liable to be modified by changed conditions of life, we may believe it possible that the various parts of a plant might be modified through the agency of its own altered secretions. With such facts before us, we need feel no surprise at the appearance of any modification in an organic being."

The removal, then, of a cell wall has no disastrous effect, but the continual removal of a plant cell wall by a larva being accompanied either by salivary excretion or by an upsetting of the metabolism of the cell or both results in the formation of galls.

The discovery has been made that by treating proliferating cells with various poisons and chemical reagents (quinine, chloral, cocaine, antipyrin, &c.), irregularities in their mitoses and mode of growth can be artificially produced, even when quite dilute solutions are used. Thus the reduction in the number of chromosomes usually associated with generative cells, and which has been also demonstrated in certain cancerous cells, may be due to the presence of some specific chemical substance.

I have not been able to convince myself as to the irregularity of mitosis and reduction in the number of chromosomes in galls, but if it exists the similarity between galls and cancer, which has often been pointed out, will be enhanced. Moreover, if a substance capable of dissolving cell walls or in any way modifying their growth exists in tumours upon plants or in tumours upon animals, then the successful grafting of tumours from one plant to another, or from one animal to another, does not necessitate the supposition that any organism must be present also.

It is not my purpose here to discuss in any way the cause of cancer, but I may be permitted to make one or two brief references.

W. Roger Williams[1] quotes numerous cases of auto-implantation, and mentions the frequency with which, after tapping for ascites due to cancerous tissue, the wound-track itself became cancerous, as if through implantation in it of cancerous elements, conveyed there by the ascitic fluid.

There are, he says, good reasons for believing that eroded surfaces may be infected through contact even with the discharge from cancerous ulcers, and many surgeons now admit the traumatic dissemination of the disease through the inoculating of wounds by the escape of " cancer juice " during operations.

According to Adamkiewiegz,[2] however, the infectivity of cancer is so well established that the parasitic nature may be regarded as a certainty. He maintains that the constituent cells of cancerous tumours are not epithelial cells, as is generally believed, but parasitic organisms : " A cancer cell is not an epithelial cell, but an animal—a protozoon—especially a coccidium (*Coccidium sarcolytes*)." Since all parasitic microbes produce toxins, Adamkiewiez believes that there must be also a special cancer toxin, and this he claims to have discovered in a substance termed by him " cancroin," which he has extracted from malignant tumours. He finds that this substance is chemically akin to neurin, tri-methyl-vinyl-ammonium hydroxide. By implanting brain substance and other tissues from cancer patients into divers regions of various animals, Adamkiewiez claims to have caused in them a special intoxication owing to the action of this toxin.

This toxin of Adamkiewiez is, however, closely allied to cholin, which, as shown in an earlier part of this paper, is the very substance Loeb considers as playing such an important part in the segmentation of his artificially fertilised sea-urchin eggs, and which is produced synchronously with segmentation, or as a secondary product of proliferation.

Neurin is vinyl-trimethyl-ammonium hydroxide, $N\ (C_2H_3)\ (CH_3)\ 3OH$; while cholin is hydroxyethyl-trimethyl-ammonium hydroxide $N\ (C_2H_4OH)\ (CH_3)\ 3OH$.

From the foregoing the following conclusions may be drawn :—

1. In the present state of our knowledge the behaviour of the living cell membrane towards the substances with which it comes in contact is, in many cases, incapable of interpretation on a purely physical basis.
2. The limiting membrane of living cells cannot be compared with dead parchment, because—
    - (1) through it the protoplasm is frequently continuous ;
    - (2) it is generally coated on the outside with a slime of a colloid nature.

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[1] *The Natural History of Cancer*, 1908, *p.* 187.

[2] *Untersuche nüber den Krebs*, Wien, 1893.

3. Colloids in particular show the phenomenon of adsorption and usually have an electrical charge upon them which influences positive or negative ions that come within its sphere of influence. The electrical charge upon a colloid may alter according as it is in an acid or alkaline solution.
4. To the slime of colloid nature external to the wall of a cell are to be attributed important functions in determining what passes into or out of the cell.
5. The cell wall is largely concerned in limiting the growth of the cell, its continual removal by mechanical means or chemical means, or both, results in rendering active the quiescent meristematic powers of the cell.

ON THE ACHSELBOGEN MUSCLE.

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(With seven figures.)

I. Summary of state of opinion.

The term "achselbogen" was first introduced by Langer, in 1846, to designate the aponeurotic, or even tendinous, arch frequently met with bridging across the axilla from the latissimus to the pectoral tendons or fascia over the coraco-brachialis muscle, and arching over the neuro-vascular bundle as it passes from the axillary fossa into the brachium. This arch, when present (*cf.* figs. II and III), forms a definite thickened margin of the axillary fascia, concave towards the brachium. Although it may be present without any associated muscular slip, yet when axillary muscular slips are present they invariably exhibit such intimate connections or relations with this fibrous band as to make it clear that it is morphologically as well as topographically associated with them. Hence Langer's term "achselbogen" has gradually come to signify, not the fibrous band specially, but also an arcuate axillary muscular slip which either replaces it wholly or partially, or is in close and intimate connection with it; in fact, the term achselbogen by itself now ordinarily denotes the muscular slip. This is, perhaps, a little unfortunate, and it will be advantageous if the expressions "muscular achselbogen" or "achselbogen muscle" be employed to designate the muscle, thus permitting the use of the term "tendinous achselbogen" to indicate the arcuate structure of Langer.

It is still an open question whether all aberrant axillary muscular slips described as achselbogen muscles are actually identical in morphological value and significance. In other words, it may be necessary to distinguish between a genuine achselbogen muscle and certain other muscular elements occasionally met with, which, in spite of a tolerably close relation to the achselbogen, are yet to be interpreted in a different sense.

The typical muscular achselbogen is a muscular band, somewhat variable in shape and extent, as well as in its precise attachments, which has long been recognised as occurring within human axilla. It ordinarily arises from the *m. latissimus dorsi,* and is usually inserted in close association with the deeper portion of the great pectoral tendon.

The earliest known mention of the muscle is in the *Edinburgh Medical Journal* for 1812, by Ramsay, who appears to have discovered it in 1795. Descriptive references to it are to be met with in the works of a number of the earlier nineteenth century anatomists. But it aroused little discussion until it became an object of that interest in morphological interpretation which was the immediate fruit of the theory of evolution after the middle of last century.

It is not my purpose to attempt to give a full account of the observations and discussion which have been carried on in regard to this muscle. For this object recourse may be had to the comparatively recent papers of Ruge (**1**, **2**, and **3**), of his pupil Tobler, and of Heiderich (**6**), and Böse (**5**). There is, indeed, already a rather voluminous literature dealing with this little muscle. Along with the sternalis muscle, it has served as the occasion for the discussion of certain significant problems of mammalian myology to a greater degree than most other muscular anomalies or varieties.*

Practically, only two views of the nature of the achselbogen muscle are now entertained. Both of these we owe originally to two British anatomists—Turner, of Edinburgh, and Humphry, of Cambridge.

A morphological interpretation of the muscle in the modern sense cannot be said to have been attempted until, in 1867, Turner, then an assiduous and acute investigator of human anatomical variations, suggested that the muscular arch might very probably represent a vestige of the *panniculus carnosus* of other mammals. A little later Humphry put forward the view, to which he had been led by his observations on the myology, more especially of submammalian forms, that such "connecting axillary bands" were the result of an "imperfect segmentation" of the pectoralis from the latissimus portion of his "superficial brachiocephalic sheet" of the ventro-appendicular muscular mass, which in certain lower forms he had found entirely unsegmented.

These views were alike based solely on the consideration of the comparative anatomy of the muscular system itself. Gradually it became recognised, in the study of comparative myology, that the innervation of a muscle was a factor of the utmost significance for the determination of muscle homologies.

Twenty-three years ago I described a case of bilateral achselbogen muscle in which I had traced the nerve supply on the one side to the intercostobrachial nerve, and on the other to the internal anterior thoracic nerve. At that time I had been unable to discover any previous record of the innervation of the muscle. Subsequently it appeared that a case of its innervation from the internal anterior thoracic nerve had been published by v. Bardeleben in 1881, and that Cunningham had communicated to the British Association meeting at Montreal in 1884 two otherwise unpublished cases of innervation of the muscle from a similar source.

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* *Cf.* especially the bibliographies of Tobler and Ruge for literature prior to 1905.

On the basis of its innervation, together with what was then known of the morphology of the achselbogen muscle, I advocated that theory of the homology of the muscle propounded by Turner—*i.e.*, that it should be interpreted as a representative of the panniculus carnosus.

It is still my opinion that the argument followed in that paper was morphologically sound.

During the considerable period intervening there has grown up, as already stated, quite an extensive literature dealing with the morphology of the achselbogen muscle. Not only have a large number of cases been recorded in which the anatomy and innervation of the muscle in its tolerably varied forms have been accurately described, but the comparative myology of the entire axillary region has been exhaustively investigated. During this period, too, the foundations, at least, of a knowledge of the ontogeny of the muscle groups involved have been well and truly laid, thanks more especially to Bardeen and Lewis.

The course of subsequent investigation has, in the main, I believe, gone to establish the position for which I contended in 1888. It is true that the apparent discrepancy between the mode of innervation on the two sides of the case referred to have seemed unintelligible to some subsequent observers who have espoused the same general view (*cf.* Tobler, *loc. cit.* **4**, p. 503); but the discussion of this question will form the subject of a separate publication.

The most important recent contributions to our knowledge of the achselbogen muscle have come from Ruge, of Zürich, and his pupils. Ruge had himself discussed the morphological character of axillary muscles in the course of his investigation of the panniculus in monotremes. Here he had been led to the conclusion that the panniculus of mammals is directly derived from the pectoral mass whose innervation it shares. At Ruge's instigation, his pupil Tobler (**4**) investigated the comparative anatomy of the primates, with special reference to the morphology of the panniculus. The results of this investigation appeared in 1902, along with an account of some interesting cases of achselbogen muscles in the human subject. Tobler's conclusions are thus summarised:—The human achselbogen muscle represents the cranial (*i.e.* preaxial) portion of the *panniculus carnosus*. The variants from the typical achselbogen are explicable as results of—(a) variations of the panniculus itself; (b) preservation of other vestiges of the latter than those usually met with; (c) intimate connection of panniculus with pectoral mass; (d) progressive transformation (*e.g.*, hypertrophy) of vestigial remains of panniculus. The homology of achselbogen with *panniculus carnosus* is confirmed by its innervation, which is from the anterior thoracic nerves.

In 1905 Ruge himself (**2**) produced a comprehensive review of the panniculus question in its relation to both sternalis and achselbogen muscles. Through the numerous sections in this critical study the author systematically examines the several hypotheses regarding the nature and structural affinities of both sternalis and achselbogen. Both, he concludes, are vestigial representatives of the panniculus.

He discusses and dismisses the interpretation of achselbogen as remains of a primary connection between pectoralis major and *latissimus dorsi.* He will not absolutely deny the occurrence of genuine musculo-tendinous connection between the pectorals and latissimus, although he doubts the adequacy of the proofs so far adduced. But any such connections are secondary, and not primary. Many cases of such alleged connecting slips are themselves mere panniculus remains, which have acquired more intimate connection with the latissimus or pectoral. Or, if in some cases they are genuine extensions of latissimus elements to the pectoral tendons, such extensions are never primary, as Humphry first supposed. The connecting link between the two muscular masses in such cases is not even to be regarded as secondarily attained through the direct extension from one to the other. It has only been effected through the agency of an intermediary muscular element in the shape of a genuine panniculus slip or true achselbogen. If, as occurs in some cases, this intermediary agent disappears, then a latissimo-pectoral arcuate connection may survive, suggesting an original continuity between them in Humphry's sense.

Such, briefly, is the general view of Ruge and his followers with reference to the morphological significance of achselbogen.

According to them, further, the subcutaneous muscle of the mammalian trunk may be represented in man by various muscular slips or sheets corresponding to each or any of the following subdivisions of the *panniculus carnosus*—(a) *pars abdominalis*; (b) *pars thoracalis lateralis*; (c) *pars axillaris et dorsalis.* The last-named includes most slips recognised as muscular achselbogen, and generally arising superficially from the *latissimus dorsi,* as well as some at least of the supposed aberrant slips of the latissimus itself. The second category includes fasciculi and thin muscular sheets occasionally met with arising from the fascia over the serratus anterior muscle, as described—*e.g.,* by Turner (*loc. cit.*). The first category includes muscular slips which often appear as if detached from the pectoralis mass, and have near affinities with occasional pectoralis quartus slips, as well as with the more normal *pars abdominalis* of the pectoralis major itself.

The views just set forth have not, however, been accepted without demur. Several recent writers, notably Böse and Heiderich (**5** and **6**), have so far adhered to Humphry's theory, inasmuch as, whilst admitting that many achselbogen are undoubtedly to be interpreted as vestiges of the panniculus, they yet contend that other axillary muscular slips occur, often in close association with a true achselbogen, but which are themselves remains or representatives of a muscular continuity between the pectoral and latissimus sheets. In a more recent paper, in 1910, Ruge (**3**) has returned to the criticism of the view represented by Böse and Heiderich. By a careful analysis of a new case of achselbogen, he attempts to show that of three portions in which, in this case, the muscle is arranged, one portion, placed intermediately between the other two, so closely associates itself with the latissimus as to suggest, to anything short of critical examination, an actual derivation from the latter muscle—

a conclusion which, in the case cited, would be fallacious. Ruge maintains that the case for the interpretation of one group of achselbogen as latissimo-pectoral has not been proved by observations such as those of Böse and Heiderich. For the critical establishment of this position, further, more exact, observations are required. Slips that on casual examination appear to belong to the latissimus may prove, as in Ruge's last case, to be of strictly panniculus derivation. Again, genuine latissimus slips, as proved by their innervation, may at first sight appear to constitute bridging axillary slips or achselbogen; but in such cases it is necessary to inquire whether they are not really continued into a tendinous achselbogen proper—*i.e.*, the fibrous representative of a true pannicular muscular slip which has disappeared as such. In the same number of the same journal, Bluntschli (**8**), another pupil of Ruge's, describes a number of cases which appear to lend support to Ruge's scepticism with regard to the occurrence of a genuine latissimus achselbogen. Bluntschli sets himself out to investigate the rôle played by the *latissimus dorsi* in the formation of achselbogen structures. He concludes that there are no unequivocal observations which must be interpreted in favour of the existence of primitive intimate connection between the pectoralis and latissimus groups.

[In the most recent number of the same periodical (July, 1911), Sicher (**9**) describes and figures another well-marked case of what appears to be an extensive achselbogen formed purely by an extension of the latissimus insertion.]

II. Records of new cases.

I propose now to place on record a number of additional cases of the occurrence of achselbogen which have come under my observation from time to time. I do not pretend that the details of these are such as to warrant any crucial decision on the questions now at issue as regards the interpretation of the varying forms of achselbogen. But several of the cases are interesting, as illustrating certain types of axillary muscles, and as such are worthy of passing commentary.

I begin with some hitherto unpublished notes on one of the cases (Case 4 in my series) formerly recorded by me—that of a female, æt. 29. I now append a figure illustrating the condition there met with. Here the achselbogen (Fig. I, 7) arose partly from the tendinous inscription between it and the *latissimus dorsi* and partly from the surface of the tendon of a closely adjacent musculo-tendinous slip 9, detached from the marginal portion of the *latissimus dorsi* muscle. This slip itself ended in a thin, narrow aponeurotic tendon which, broadening out, passed to be inserted into the fascia over the coracobrachialis muscle. This slip, though detached from the marginal portion of the *latissimus dorsi* did not form the actual free border of the latissimus muscular mass. This was constituted by yet another detached slip 8, somewhat larger, but of similar character, ending also in an aponeurotic expansion 6 upon the coracobrachialis, but more proximally, nearer the coracoid 4. This aponeurotic tendon was continuous in arcuate fashion with the distal edge of the tendon of the lesser pectoral muscle 3. A considerable portion 5 of the latter tendon passed over the surface of the coracoid towards the shoulder joint.

In this case it will be observed that a true and typical achselbogen muscle coexisted with two apparent latissimus slips, and was to all appearances quite independent of the larger of these. Unfortunately, the innervation of these latissimus slips was not ascertained, though my notes indicate that no branch from the source of supply to the achselbogen could be traced to them.

In view of Ruge's recent observations, it would be necessary actually to verify the probable innervation of these slips from the thoraco-dorsal nerve before one could pronounce them to be morphologically distinct from the achselbogen. But the complete structural independence of the main marginal slip from the true aschelbogen strongly supports its character as a true latissimus element connecting with the pectoralis sheet.

Case No. 5.—Male subject, æt. 20.

In this case no achselbogen muscle was present, but there was a well-marked tendinous achselbogen (in Langer's sense—see Figs. II and III). Fig. II illustrates the condition as visible in the earlier stage of the dissection on the right side. The acute tendinous arch was continuous by its convexity with the latissimus dorsally, with pectoral fasciculi ventrally, as well as with the fascia covering these and the serratus anterior muscle. Its concavity was continued into a thin fascia (not shown in the figure), which helped to close in the fossa axillaris.

A thin, flattened bundle of muscular fibres, 15-20 mm. in width, parallel with and close to the border of the pectoralis major, was inserted with the tendinous achselbogen, and was throughout its course intimately connected with the dense axillary fascia overlying it, and through which it was visible.

On reflecting the pectoralis major an additional muscular slip (Fig. III, 3) was found under cover of its lower border, blended with the latter at their origin, but separating from the pectoralis to be inserted into the ventral horn of the tendinous achselbogen. This latter was deeply continued into the fascia over the coracoid muscles.

On the left side the condition was similar, but the second (deeper) pectoral slip was absent. On the other hand, there was on this side a separate fasciculus, detached from the border of the *latissimus dorsi*, and gradually diverging from the latter, to be inserted into the dorsal horn of the tendinous achselbogen.

On both sides a few tendinous fibres representing a dorso-epitrochlear slip, passed from the latissimus tendon to the long head of the triceps brachii.

The aberrant pectoral fasciculi above-described are no doubt homologous to the pectoralis quartus slips so often described. The entire arrangement may be usefully compared with, *e.g.*, Tobler's figs. 23 and 24, and Bluntschli's figs. 2, 3, 4, and 6; compare also Heiderich's figs. 7, 9, 10, and 12.

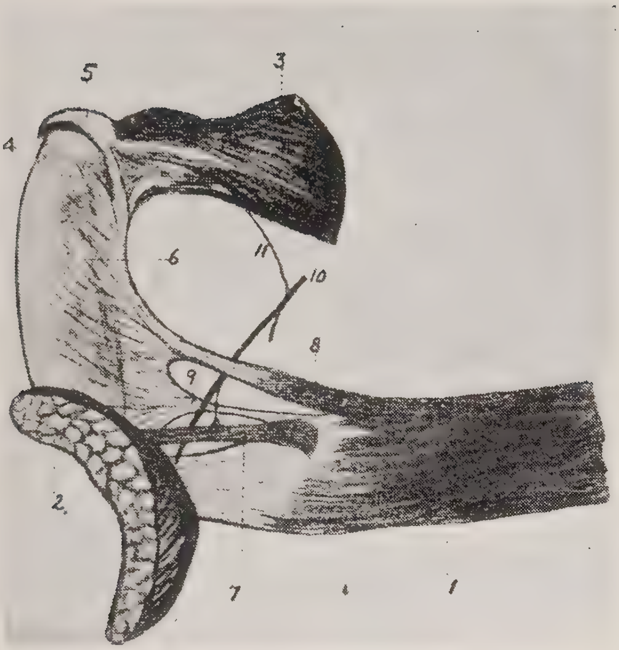

Fig. I—Case IV.

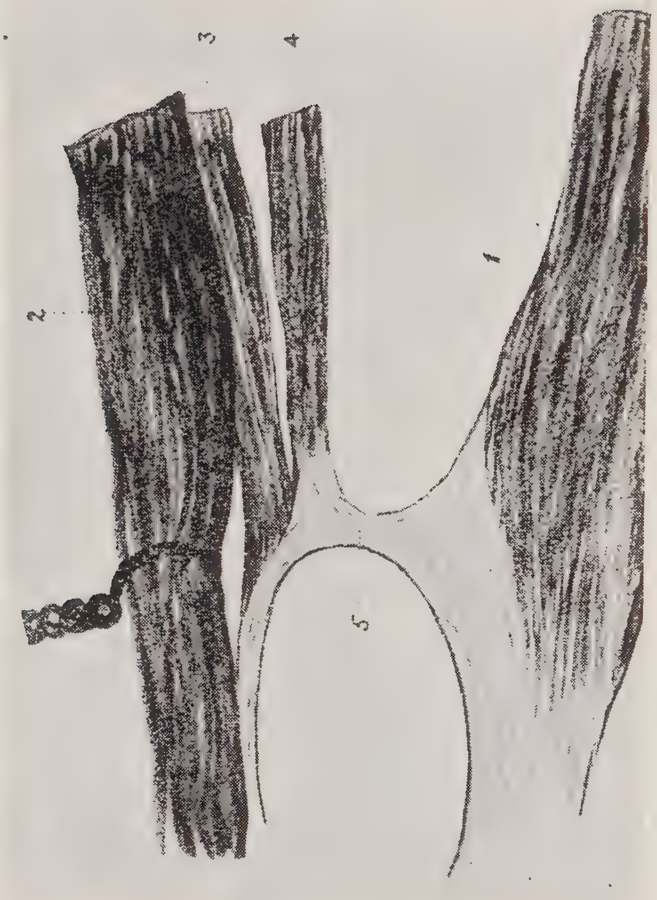

Fig. III—Case V.

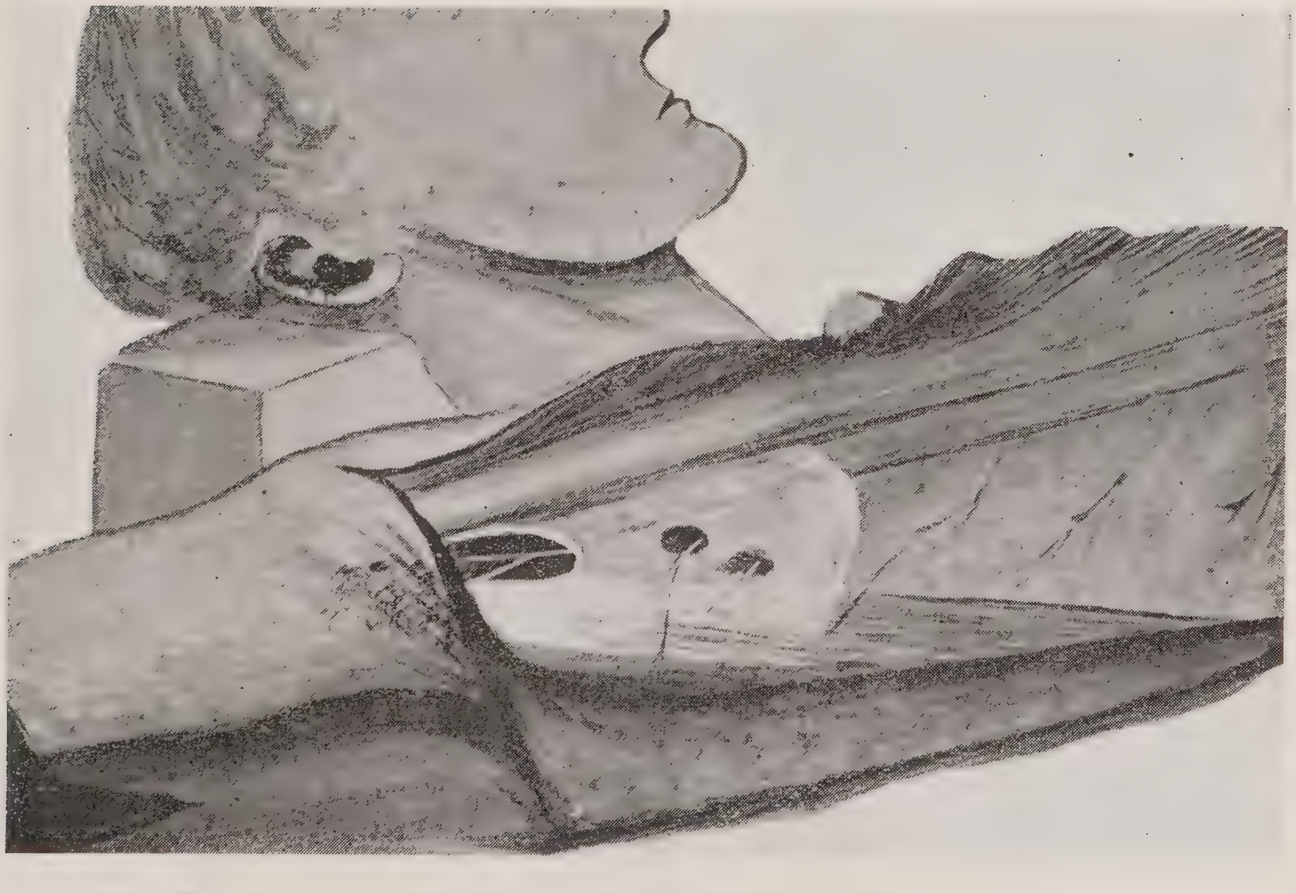

Fig. II—Case V.

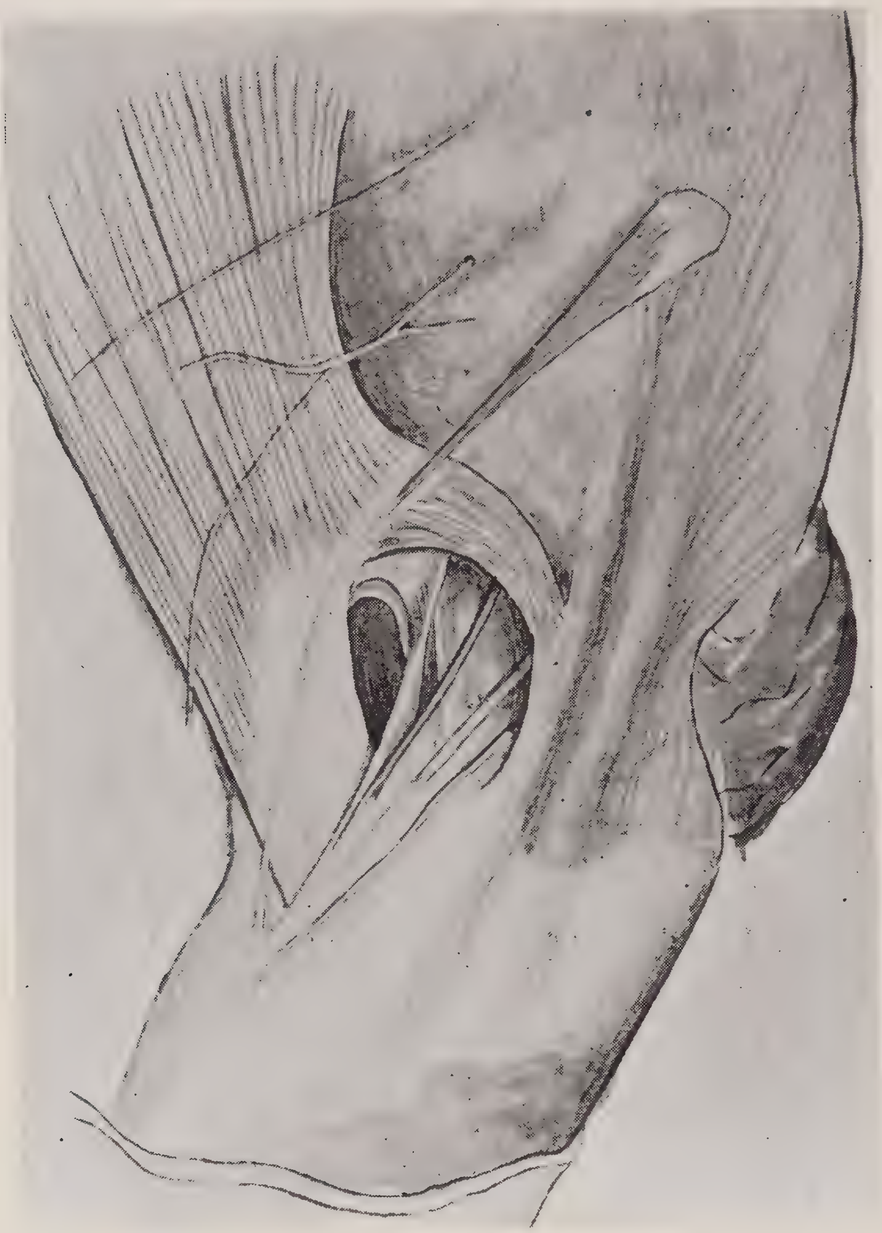

Fig. IV—Case VIII.

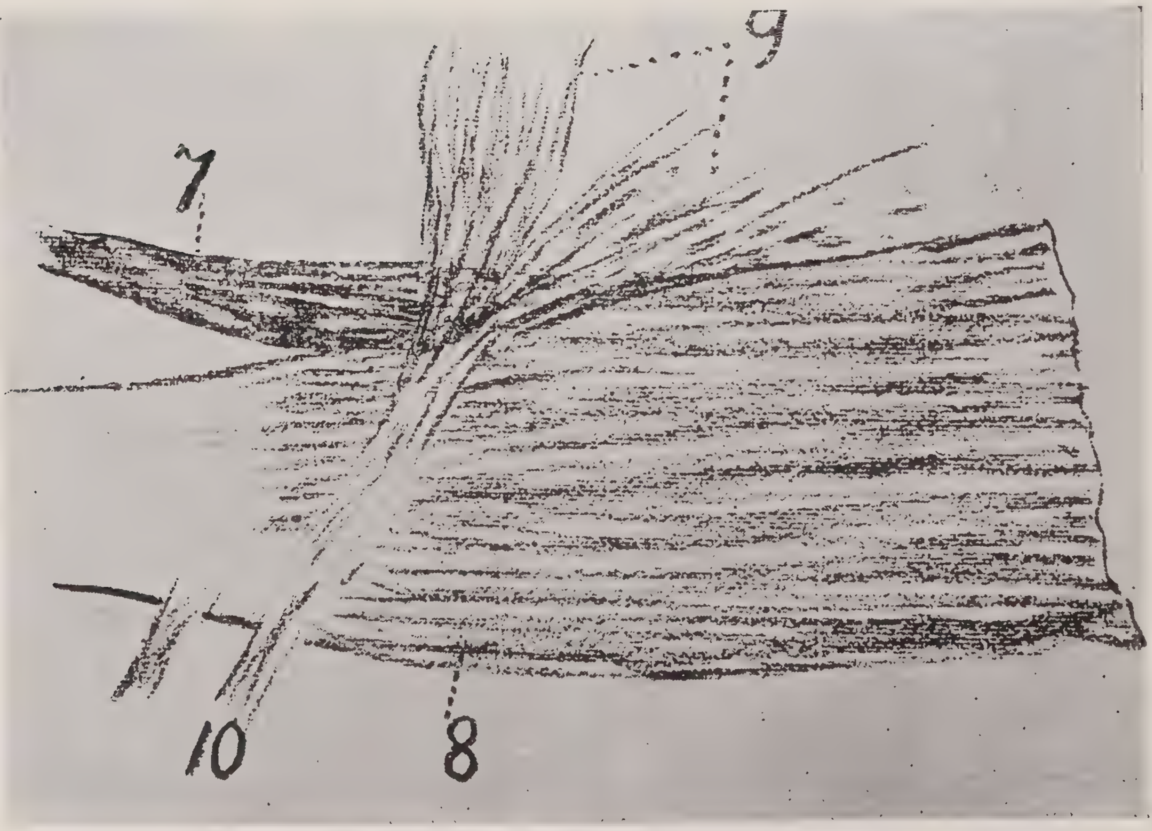

Fig V—Case IX.

CASE NO. 8,* Fig. IV.

On the left side of the body of an elderly (British) male subject, the *latissimus dorsi*, as it approached the axilla, was seen to be much broader than usual, its anterior or axillary border thinning out and extending across the axilla beneath the deep fascia so as to cover in a large part of the floor of the axillary fossa. The fibres of this anterior thin expanded part of the muscle ran parallel to those of the rest of the muscle, and ended in a transverse tendinous band, which abruptly terminated their course, but which gave origin on its other (brachial) aspect to the fleshy fibres of a broad achselbogen, which crossed the large vessels, to end in an expansion of the great pectoral tendon. The tendinous inscription, however, was also the site of insertion of another muscular slip. This arose by a wide aponeurotic origin of scattered muscular fibres over the inferior portion of the anterior surface of the pectoralis major, and, narrowing gradually into a thin, flat, muscular band, it turned round the lower rounded border of the pectoralis major, and then passed onwards, to be inserted into the superficial aspect of the inscription in a direction at right-angles to that of the fibres between which the inscription was placed. Further, a few strong aponeurotic fibres continued the course of the tendinous inscription distally in the arm, crossing the superficial aspect of the *latissimus dorsi* tendon to join the triceps as a " dorso-epitrochlearis " band.

The achselbogen, with the thin, muscular sheet from the surface of the pectoralis major, were both supplied by two long delicate twigs coming off by a common slender stem from the internal anterior thoracic nerve.

An achselbogen was also present on the right side, but its source of innervation was not ascertained.

CASE NO. 9.

In a muscular Chinese subject, æt. 47, achselbogen were present in both axillæ, and were very distinctly visible in the intact body.

On the right side a strongly-developed achselbogen (Fig. V, 7) extended from the tendinous inscription between it and the axillary border of the *latissimus dorsi*, to join the lowest part of the tendon of the great pectoral.

Superficial to the origin of this slip, however, was a layer of muscular fibres 9 inserted into an aponeurotic band, which was directed obliquely across the surface of the *latissimus dorsi* 8 near the junction of its fleshy and tendinous portions. This aponeurotic band was continued downwards into the triceps, forming a dorso-epitrochearis slip 10. The fleshy fibres forming this thin, superficial, muscular layer converged to their aponeurotic insertion from different directions. Many were apparently derived from the superficial part of the axillary border of the latissimus itself; others simply arose from the axillary fascia; whilst a broad and definite band of muscular fibres arose ventrally from a strong pectoral fascia over the superficial aspect of the pectoralis major. This band was about an inch and a half broad at its transition into its aponeurosis of insertion, which was superficially adherent to the latissimus-achselbogen tendinous inscription.

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* The numbers are the serial numbers of individual bodies in which the writer has traced the innervation of an achselbogen on one or on both sides.

I would draw special attention to the continuation, in both of the cases Nos. 8 and 9, of the tendon of the superficial pectoral slip, as a dorsi-epitrochlearis prolongation, into the fascia over the long head of the triceps brachii muscle. A similar condition is reported by Cunningham (p. 384) as follows:—"A long tendinous dorsi-epitrochlearis was present, and it took origin, not from the *latissimus dorsi,* but from the tendon of the achselbogen."

In the cases above described (Nos. 8 and 9), the dorsi-epitrochlear slip was not only adherent both to the latissimus tendon and to the tendinous origin of the achselbogen proper, but was the direct continuation of the superficial pectoral slip. The condition of the superficial pectoral slip shown in Gehry's fig. (10) is obviously illustrative of a fundamentally similar arrangement, which must be of profound significance for the morphology of the condition.

The nerve supply of this rather extensive superficial muscular sheet was derived from a considerable branch from the *anterior thoracic plexus—i.e.*, the ansa between the external and internal thoracic nerves, which was plexiform in character.

The deeper muscular slip or true achselbogen did not appear to be innervated from this source at all, or at least not directly. It appeared to receive its supply through a large branch of the intercostobrachial nerve, which passed downwards to pierce it, and which then turned upwards to communicate with those twigs of the thoracic nerves supplying the superficial sheet of muscle. It is highly probable that in this communication there was effected an exchange of filaments between the two nerves, and that here we have to deal with a case of double nerve supply to the achselbogen. There can be little doubt that the intercostobrachial nerve was concerned either exclusively, or, more probably, partially, in the supply of the muscle.

On the left side the whole condition was substantially the same, but the superficial structure of muscular fibres converging to the tendon of the *latissimus dorsi* was more definite and extensive, and a layer of sparse fibres arose from the fascia covering the serratus anterior muscle, so that we may say that fibres converged from the anterior and lateral aspects of the chest to form the sheet in question and its terminal part on both sides had a superficial relation to the intersection between latissimus and achselbogen proper.

The achselbogen on this side was supplied definitely by branches from the anterior thoracic plexus, and there was no branch of the intercostobrachial piercing the muscle and hooking back again to communicate with the anterior thoracic nerve supply.

Case No. 10.—Male, æt. 25.

Achselbogen broad, thick, and flat, visible in both axillæ in untouched body.

Both arose from *latissimus dorsi* by an extensive tendinous inscription. The *latissimus dorsi* extended ventrally as a thin sheet of fleshy fibres as far as mid-axillary line, but the fibres of this ventral extension were continuous with the rest of the muscle and with its true tendon, and nerve fibres from the long subscapular thoracodorsal nerve were traced to them.

On each side the achselbogen was supplied by a branch of the internal anterior thoracic nerve, but a large bundle of fibres was continued from this nerve of supply under cover of the achselbogen down to the ventro-medial aspect of the upper arm as a second nerve of Wrisberg, the primary representative of which nerve was present in its usual position close to the axillary vein.

The intercostobrachial nerve on each side was comparatively small.

Case No. 11.—Male, æt. 74.

Muscles exceedingly well developed. Achselbogen in left axilla.

The *latissimus dorsi* reached, ventrally, as far as the mid-axillary line, the ventral 50 mm. of its axillary sector forming a thin fleshy layer, ending in an arcuate tendon which bounded an oval opening into the axillary fossa, and was continued, partly fleshy and partly as a thin aponeurotic expansion, on to the surface of the coracobrachialis muscle. From the concavity of the tendinous arch so formed arose a true achselbogen, short, but thick and fleshy. This ended in a manner similar to the ending of the latissimus slip, upon the coracobrachialis muscle, but more distally, its thin tendon being lost upon that muscle. But its thin tendon was overlapped by yet another aponeurotic expansion, which formed the tendon of insertion of a *pectoralis quartus* band, about 30 mm. in width. This arose from the aponeurosis of the external oblique muscle at the level of the sixth rib, and extended, laterally, parallel with the pectoralis major, and slightly overlapped by its caudal border. The origin of the pectoralis major was from the five costal arches only, and not from the aponeurosis of the external oblique at all; but it was, nevertheless, very strong and fleshy, and otherwise well-developed. It was separated from the *pectoralis quartus* slip by the exit of the ventral ramus of the lateral cutaneous branch of the fourth intercostal nerve, so that the separation of the slip from the other pectoralis was a genuine segmentation. The *latissimus dorsi* arose by its thin anterior expanded portion from the eighth and ninth, as well as from the tenth, eleventh, and twelfth ribs, and the digitation of the serratus anterior from the eighth rib accordingly consisted of only a few fibres.

The achselbogen was innervated by a long slender branch from the internal anterior thoracic nerve. Coming off the latter quite close to the nerve to the achselbogen was a similar long slender branch supplying the *pectoralis quartus* But the musculo-tendinous arched slip of the *latissimus dorsi* (a spurious achselbogen) was continuous with the rest of the *latissimus dorsi*, and was distinctly supplied by a branch of the long subscapular (thoraco-dorsal) nerve to that part of the muscle.

It is to be observed that in both the cases 10 and 11 the ventral axillary extension of the latissimus sheet was proved to be innervated from the thoraco-dorsal nerve, whilst the true achselbogen, which were also present, were innervated by the anterior thoracic nerves.

In case 11 there can be no doubt of the genuine latissimus character of the slip which joined the coraco-brachial fascia distal to the achselbogen. Still, Ruge's contention that in such a case the true or panniculus-achselbogen is really responsible for the latissimus prolongation may be quite legitimate, and is perhaps more probable than the view that it represents a relic of a primary connection of the pectoral and latissimus sheets.

CASE NO. 12 - Male, æt. 46

True achselbogen in both axillæ.

Each arose from the brachial side of tendinous inscription between it and the *latissimus dorsi.*

On the right side the innervation of the muscle was double. One filament entered it from the internal anterior thoracic nerve, but, in addition, it was supplied by numerous large filaments from the intercostobrachial nerve. Previous to this, however, the intercostobrachial had exchanged fibres through an ansal communication with the internal anterior thoracic nerve from which there was no separate nerve of Wrisberg.

On the left side the arrangement was somewhat similar. The achselbogen had the same double nerve supply; but here, although the intercostobrachial nerve had a communicating loop with the internal anterior thoracic nerve as on the other side, some of the filaments of the former entering the muscle could be traced up into the intercostobrachial trunk above the level of communication between it and the internal anterior thoracic nerve. The latter communication was, on this side, double and decussating.

CASE NO. 13

Large achselbogen (on the left side only).

Arises in ordinary manner by a broad origin, about 50 mm. wide, from the outer surface of the *latissimus dorsi,* from whose substance it is separated by a tendinous inscription.

Its origin is pierced by lateral cutaneous branch of third intercostal nerve.

It is inserted into a very broad triangular and well-defined aponeurosis, whose distal part is fused to the deeper aspect of the tendon of the pectoralis major, and whose proximal part, which completely hides the *m. coraco-brachialis,* extends up to the coracoid process, where it joins the insertion of the pectoralis minor.

The nerve supply is from a large branch from the internal anterior thoracic nerve which enters the muscle near its origin.

CASE NO. 14.

In this case a typical achselbogen was present on the right side of the body (Fig. VI). It arose in the usual way by a tendinous inscription from the *latissimus dorsi.* It joined an aponeurotic expansion under the greater pectoral, and continued up to the coracoid. This expansion was also joined by a *pectoralis quartus* slip which was also present. The nerve supply of the achselbogen was double, its main nerve supply came from the internal anterior thoracic nerve, and soon after its origin gave off a slender twig which supplied the *pectoralis quartus* and communicated in the substance of the greater pectoral muscle with another branch of the internal anterior thoracic

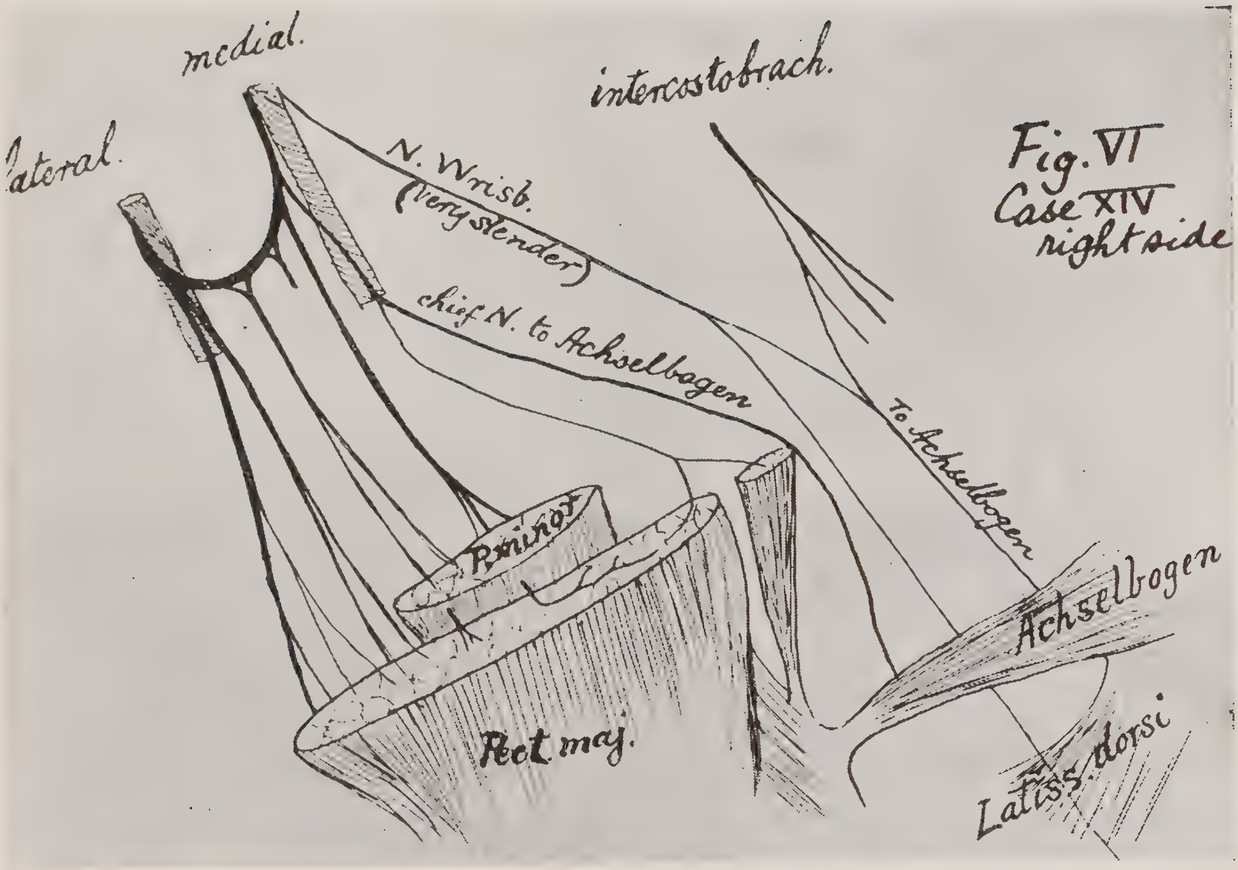

Fig. VI—Case XIV. Right Side.

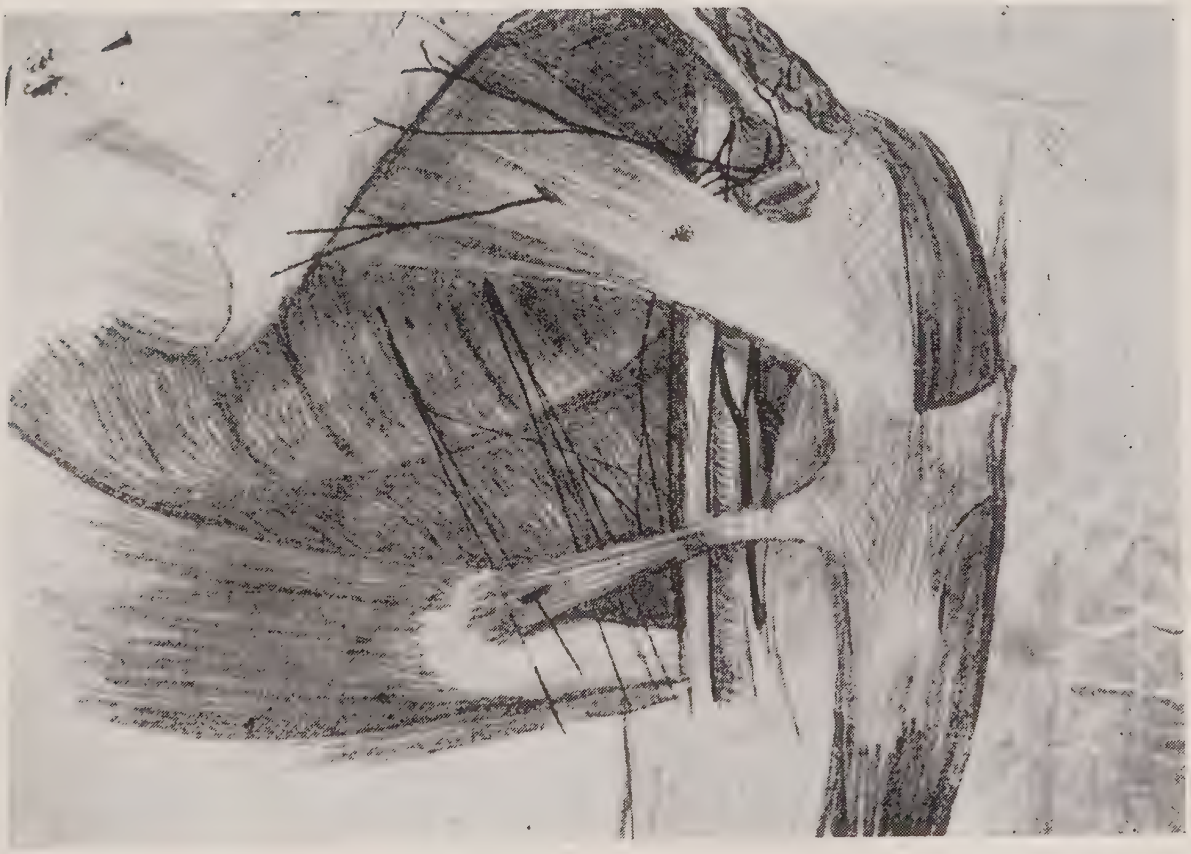

Fig. VII—Case XIV.

nerve to that muscle. The second nerve supply to the achselbogen was formed by two components; both of these were slender filaments, one coming from the nerve of Wrisberg (which was itself extremely small), and the other was a branch of the intercostrabrachial. These two filaments joined at an acute angle, and the nerve so formed was continued bodily into the achselbogen not far from its latissimus attachment.

On the left side of the same subject the condition was somewhat similar in character, except that the *pectoralis quartus* slip was absent (Fig. VII). There was only one nerve of supply to the achselbogen, and this consisted of two components joining at an acute angle, one from a branch of the internal anterior thoracic, the other from a branch of the intercostohumeral. On this side a branch of the ramus lateralis of the third intercostal nerve pierced the achselbogen near its origin, though it gave off no branch to the muscle. It communicated, however, by an independent filament with the nerve of Wrisberg.

CASE No. 15.

Left side.—Short, stout, triangular or conical muscle passing by an elongated aponeurotic tendon into the subpectoral fascia. The muscle had a double nerve supply—(a) by a small twig from the intercostobrachial nerve; (b) by a lairly large nerve emerging as a ramus lateralis from the first intercostal space in series with the other rami laterales. The two nerves did not communicate before entering the muscle, and the ramus lateralis of the first intercostal gave off no cutaneous branch to the brachium.

CASE No. 16.

Left side.—Triangular achselbogen about 25 mm. broad and 50 mm. long, arising by tendinous inscription from the *latissimus dorsi*, superficially. Some deeper fibres, however, could be traced along the anterior border of the latissimus, with which muscle they apparently became continuous. It was inserted by a long, narrow tendon which expanded under cover of the greater pectoral. The lower part of the expansion fused with the deep aspect of the pectoralis, and thus gained insertion into the humerus and the deep fascia of the arm. The upper part of the expansion was more independent of the greater pectoral tendon, from which it received an accession of fibres. It curved upwards, crossing the sulcus inter-tubercularis, to be attached to the crest of the greater tubercle. The nerve supply was derived from the ramus lateralis of the third intercostal nerve by a well-marked branch which entered the anterior border of the muscle. This ramus lateralis of the third intercostal nerve was large, and crossed the axilla in two large trunks. The lower, more caudal, of the two trunks supplied the nerve to the achselbogen, and was then continued onwards to the integument. The upper trunk passed under the axillary arch, and was distributed to the medio-dorsal aspect of the upper arm, extending as far as the elbow. The intercostobrachial nerve of the second intercostal was present, smaller than usual and restricted in its distribution, though still extending as far as the distal third of the brachium. Further dissection within the thorax failed to reveal any connection between the ventral primary divisions of the second and third intercostal nerves.

Right Side.—An achselbogen was present on this side also. At its origin from the *latissimus dorsi* the tendinous inscription entirely cut it off from muscular continuity with the latter muscle. It formed a small oblong muscle about 9 mm. broad and 35 mm. long. Its aponeurotic expansion over the biceps and coraco-brachialis muscles reached as high as the corocoid process. This tendinous expansion also received on this side the insertion of a *pectoralis quartus.* The nerve of supply came from the internal anterior thoracic nerve, and the exceptional development of the rami laterales of the second and third intercostal nerves did not occur on this side.

CASE NO. 17.

Achselbogen was present on both sides. On the right side it was small, and its connections had been interfered with before it was observed. The left muscle arose by two heads—one dorsally and superficially from the surface of the *latissimus dorsi* at the junction of its muscular and tendinous portions, the other head of origin was a long, slender band of muscular fibres running along the anterior border of the *latissimus dorsi*, but apparently independent of that muscle and separated from it by a strip of areolar tissue. More caudally the fasciculi seemed to arise independently by scattered bundles, interspersed with areolar tissue, from the fascia covering the serratus anterior. This anterior slip of origin (*pars thoracalis lateralis*) joined the other slip about 12 mm. from the origin of the latter, but, before doing so, it gave off one of the two tendinous bands which formed the insertion of the muscle. This was a slender tendino-aponeurotic band, which ran up into the axilla under cover of the pectoralis major to gain an insertion into the conjoined tendon of the biceps and coraco-brachialis. The main muscular belly of the achselbogen ended in a narrow tendon which spread out into a thin expansion under cover of the greater pectoral tendon, and, fusing with this, gained the pectoral ridge of the humerous. The nerve of supply was found, but unfortunately its connection was temporarily lost. Further dissection revealed what was certainly the cut end of the nerve of supply which came from the internal anterior thoracic nerve. The intercostobrachial nerve pierced the muscle, but did not supply it. In this case the nerve of Wrisberg, the intercostobrachial nerve, and the rami laterales of the third and fourth intercostal nerves formed a series of loops of communication, thus constituting a simple but definite plexus along the side of the chest wall.

CASE NO. 18.—Microcephalic, female subject, æt. 53.

Double-sided achselbogen. On the right side the nerve supply was unascertained. The left side the nerve supply was mainly from a ramus lateralis of the first intercostal nerve, with a delicate communication with the internal anterior thoracic, and on the other hand double communication with the intercostobrachial. The detailed nerve arrangements in this case will be dealt with *in extenso* in a further publication.

CASE NO. 19.—Elderly male subject.

Achselbogen present on left side only. Nerve supply from internal anterior thoracic nerve.

CASE NO. 20.—Elderly male subject.

Achselbogen present on right side only. This muscle consisted of two laminæ corresponding to Ruge's (3) dorsal and intermediate laminæ respectively. In close association with the muscle there was also a dorso-epitrochlearis slip arising from the tendon of the *latissimus dorsi* close to the tendinous inscription of the achselbogen, and passing to be inserted into the brachial aponeurosis. The nerve supply to the achselbogen was from the internal anterior thoracic nerve; the nerve pierced the aponeurotic lamina of the intermediate achselbogen layer to reach the fleshy belly of the muscle.

III. Commentary.

The weight of opinion at the present time is strongly in favour of Turner's original view that the achselbogen muscle is a vestige of the *panniculus carnosus.*

But whilst this view may be said to be practically accepted as regards what may be called the true (hence the panniculus) achselbogen, it is by no means unanimously admitted that all axillary muscular slips are of identical or even similar character.

Putting aside axillary slips, obviously detached from the pectoralis mass, including the *pectoralis quartus,* there are other axillary muscular slips closely associated with the latissimus, and distinct from, and often co-existing with, true achselbogen, whose morphological value cannot yet be said to be definitely settled.

Those which are not only continuous with the *latissimus dorsi,* but are, like it, innervated by the thoraco-dorsal nerve, may be confidently reckoned to the latissimus mass. Even in these cases, however, Ruge maintains that their segregation from the rest of the latissimus is due to their original association with a panniculus element, even where the latter may have itself become suppressed.

All other axillary slips, no matter how closely associated with the latissimus, if innervated from anterior thoracic nerves, are held by Ruge and his followers to be, fundamentally, panniculus elements.

Whilst on the whole inclining towards Ruge's views, I cannot help feeling that he is inclined to press the doctrine of nerve-muscle correlation too far. It may be admitted that we have no single criterion of muscle homology of comparable value to that of innervation; but I nevertheless think that it may fairly be urged that it is by no means certain that the same muscle-fibre-groups in one embryo are invariably neuralised by precisely the same groups of axones in all other embryos of the same species.

The facts of *pre-* and *post-* fixation of the limb-plexuses speak for a directly contrary conclusion, and, heretical though it may be to say so, I see no reason to believe that there may not be a similar degree of individual variability in the dorso-ventral innervation-fields of the motor constituents of the nerves of the limb plexuses.

In a paper to be published elsewhere, I hope to demonstrate, as against the hard-and-fast innervation doctrine of Ruge, that at least the innervation path to the achselbogen muscle is not by any means so invariable as he has consistently maintained it to be.

Finally, I desire to record the conviction that a complete understanding of the axillary musculature will be found to involve an explanation of the continuity so commonly observed between dorso-épitrochlear bands and such axillary muscular slips as are described above in Cases 8 and 9, and illustrated in Figs IV and V. Any interpretation which ignores, or inadequately explains, this very definite, significant and frequently occurring connection must be a faulty one.

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ANTHROPOMETRY OF THE HUMAN EAR.

A Preliminary Communication.

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IF any excuse is needed for an investigation on the Caucasian ear, it is furnished by Duckworth,[1] who writes that " the racial aspect of the anthropology of the external ear still awaits investigation capable of leading to definite general conclusions." To gain this we must have firmly established a type ear for the Caucasian to be used as a ground for comparison. That this type has not yet been reached is easily demonstrable. Thus Berry[2] writes: " that the tendency of the modern European female to possess a higher morphological ear index than the male is a sign of marked and high civilization and of advancement "; while Rohrer[3] states that the " human auricle is in a state of degenerate metamorphosis," such retrogression having proceeded further in the female, whose ear " is now more designed for elegance and beauty than for acoustical or protective function." These two anthropologists differ radically as to what changes in the ear mean advancement and what retrogression, and till there is some agreement on points such as these it is impossible to draw conclusions from a racial aspect.

Object.

The object of the present research is to endeavour to clear away some of the conflicting statements which have been made with regard to ear diameters, and to test the usefulness of certain of these measurements, particularly the " Morphological Ear Index " of Schwalbe, quoted by Bardeleben.[4]

Material.

As this is merely a preliminary communication, the numbers as yet available are small, only fifty ears having been examined to date, the ear in each case being the left one. The cases examined were students, for the most part medical students of the University of Melbourne, and I take this opportunity of tendering them my thanks for their invaluable assistance.

Their ages ranged from 19 to 30 years, and at the outset an objection may be raised to this choice of material, for Keith[5] says that before 20 the ear " has not attained its adult form and size." Schwalbe agrees in this. The objection is certainly one that has to be admitted, and unfortunately it is one which cannot be met, but as the majority of my cases were over 20 years of age, any error due to age must be almost infinitesimal and quite negligible.

Measurements Recorded.

1. Length of ear basis.
2. Greatest length of ear.
3. Greatest breadth of ear.
4. Distance from the Darwinian tubercle to the upper border of the tragus. This corresponds to the true ear length of Schwalbe, quoted by Bardeleben.
5. Distance from the highest point of the ear to the bottom of the incisura intertragica.
6. Length of the lobule of the ear.

These six diameters were calliper measurements taken by contact only, and the procedure laid down by the British Association Anthropological Committee[6] was followed in obtaining them.

The auriculo-temporal angle of projection of Frigerio,[7] quoted by Havelock Ellis,[8] was not used, as up to the present it has only been employed in reference to criminology, but indications point to its being a valuable criterion from the racial aspect.

7. Total height of the body. Writing on the ear length, Karutz[9] shows that if people be grouped according to ear length only, the result is that tall races have long ears, and *vice versâ.* He maintains that the relative ear length, *i.e.*, the proportion of the absolute length of the ear to the bodily height is much more instructive, and the bodily height has been measured in this research to enable his proportion to be worked out.
8. Holl[10] has devoted a large amount of attention to the position of the ear on the head.

Quoting Berry[2] on this subject, we find "that Langer has pointed out that the only correct determination of the position of the ear on the head is the position of the meatus acusticus externus and its positional relation to the vertical dimensions. This usually falls in the middle of a line connecting the vertex of the head with the angulus mandibulae, the projection being on a vertical plane." In view of this work by Langer, measurements were taken—

(a) from vertex to angulus;

(b) from vertex to meatus.

The indices used are as follows:—

Aural Index (Duckworth[1]).

$$\frac{\text{Breadth of Auricle} \times 100.}{\text{Height of Auricle.}}$$

Schwalbe's Morphological Ear Index—

$$\frac{\text{Ear base} \times 100.}{\text{True ear length.}}$$

Schwalbe's Ear Modulus (Bardeleben[4]).—

$$\frac{\text{Length of Auricle} + \text{Breadth of Auricle.}}{2}$$

Proportionate ear length, Karutz.[9]

None of these foregoing characters or indices call for comment except diameter No. 4, the true ear length of Schwalbe. One definition of this diameter has already been given, and Schwalbe in Bardeleben's Handbook[4] defines it as "die Ent fernung der Darwinschen Spitze von der incisura auris anterior." It is therefore absolutely dependent on the presence of Darwin's tubercle, and that this is not always definable is universally admitted. Schwalbe[4] states it is present in 73·4 per cent. of male cases, and 32·8 per cent. of female cases. Poirier and Charpey[11] write as follows:—"Chez les Alsaciens du moins, sa présence est de régle chez l'homme et son absence chez la femme." In "Quain's Anatomy[12]" the tubercle is said to occur "in about 30 per cent. of cases examined." Testut[13] speaks of the tubercle as "abnormal chez l'homme"; while Cunningham[14] and Piersol[15] both note its greater prevalence in the male than the female, but regard it as being far from a regular feature. Keith's[4] classification of the various types of the tubercle has been accepted for the purposes of this paper, namely:—

1. A distinct triangular projection.
2. Distinct, but not pointed.
3. A distinct tubercle.

The low figure given by Quain above may be due to the fact that the less distinct forms of tubercle were not admitted, and so are not included in the 30 per cent. quoted.

The absence of the tubercle in a number of cases, as has been shown, is of grave import, because it renders the morphological index inapplicable in a large percentage of cases, ranging from 26·6 per cent. to 70 per cent. This is to be greatly regretted, because this index must, from its composition, be of extreme value in racial and sexual comparisons. Notwithstanding the literature, I resolved to obtain a morphological index in every case, even when I was not absolutely sure of the presence of a definite tubercle. When no definite tubercle was present, a point was selected which on close inspection seemed to indicate where the tubercle should be, and the true ear length was measured from such a point. If the index thus obtained varied within normal limits, it could be stated as the true ear length, and so the index derived from it could be obtained, even when no definite tubercle could be found. If the reverse were the case, then the morphological index can only be obtained in the presence of a definite tubercle, thus greatly limiting its range of usefulness. My results on this point are dealt with elsewhere. The presence or absence of the tubercle also throws light on the question of change in the ear, for if the tubercle is to be regarded as the true apex of the ear, homologous with the tip as seen in such animals as the rabbit (as has been several times affirmed, and notably so by Schwalbe[4]), then its more frequent occurrence in the male sex would point very strongly to the greater change in the female ear from the simian type as is advocated by Keith.[5] Whether this is an advance or a retrogression it is difficult to say, but the last observer's arguments as to its being a retrogression certainly seem to my mind unanswerable at our present stage of knowledge.

All the foregoing measurements have been described elsewhere, but, in addition, I have endeavoured to express numerically the degree of inrolling of the helix. This question of inrolling has been discussed by other observers, notably Keith[5] and Schwalbe,[4] but so far as I know, no attempt has been made up to the present to express it other than qualitatively; and this, even though as Keith[6] states, " exact measurements have this one supreme advantage—that they are definite facts which can be added to, verified, and compared by workers all the world over; they are not expressions of opinion or indefinite expressions of fact, which are useless to other observers." At first measurements were taken in the region of Darwin's tubercle, but the difficulty in always locating this point, which has already been referred to, rendered this line of work untenable. I then determined to obtain in each case the maximum degree of inrolling of the helix, measured from medial margin to periphery of pinna, wherever it might occur, and at the same time to note the maximum diameter of the antihelix from the margin of the cymba conchae to the margin of the pinna. Both these measurements were contact only, and were obtained by callipers. An index, which may be called the helical index, was then obtained as follows:—

$$\text{Helical Index} = \frac{\text{Maximum degree of inrolling of helix} \times 100.}{\text{Maximum diameter of anti-helix.}}$$

The measurements recorded can be summarised as follows:—

1. Length of ear basis.
2. Maximum length.
3. Maximum breadth.
4. True ear length.
5. Distance from highest point to bottom of incisura intertragica.
6. Length of lobule.
7. Total body height.
8. Position of ear—
    (a) Distance from vertex to angulus mandibulæ.
    (b) Distance from vertex to meatus acusticus externus.

From these measurements the following indices have been calculated:—

1. Aural or physiognomical index.
2. Morphological index.
3. Ear modulus.
4. Proportionate ear length.
5. Helical index.

Owing to the comparatively few cases examined to date, it does not seem advisable to go at any great length into the racial differences of the various diameters of the ear. These can be obtained from the articles by Schwalbe[4] and Berry,[2] and will be fully considered in a later communication. For the sake of comparison, however, figures relating to the Australian aboriginal ear are inserted.

Greatest Length of Ear.

Schwalbe[4] gives the following figures for this diameter in the adult male: 50·82 mm., with an average of 65·5 mm. for the left ear.

Berry,[2] for thirty male Australian aboriginals, gives an average of 65·5.

My own figures vary from 79 mm. to 56 mm., with an average of 62·8 mm.

Greatest Breadth of Ear.

Quoting Schwalbe again, we find an average of 39·2 mm., with a variation between 32 mm. and 53 mm. The Australian aboriginal ear is given by Berry[2] as having an average breadth of 34·1, while I found the breadth to vary from 27 mm. to 40 mm., with an average of 32·8 mm.

In both these diameters my figures are smaller, and notably so in the case of the breadth, than those given by Schwalbe. How much of this difference is due to the young age of my cases it is impossible to estimate, but the difference would seem to further strengthen the opinion often expressed that the size of the ear increases with advancing age.

Ear Modulus.

By means of this expression an excellent criterion is obtained for indicating the surface extent of the ear.

Using the averages given above, we obtain the following:—

From Schwalbe's figures, 52·3.

From Berry's aboriginals, 49·8.

My own figures vary from 41 to 54, with an average of 47·8. This agrees very closely with the figure, 48·6, given by Topinard[16] for eight Europeans.

Collecting together these figures which deal with the surface extent of the ear, we obtain the following table:—

	Average length.	Average breadth.	Ear modulus.
Schwalbe	65·5	39·2	52·3
Berry (Australian) ..	65·5	34·1	49·8
Anderson	62·8	32·8	47·8

Aural Index.

This is given by Schwalbe as having an average of 60·5, by Berry as 52, and my own figures work out at 52·5.

Length of lobule, position of ear on skull, and distance from the highest point to incisura tragica.

Up to the present I have been unable to work out my own figures on these points, and must defer their discussion till a later date.

True ear-length and Morphological Index.

Some attention has previously been directed to these measurements in connection with the presence or absence of Darwin's tubercle.

Schwalbe states that the morphological index varies from 83 to 195, and Frijerio gives an average of 175·3 in the same; and, bearing in mind the opinions already expressed, I expected to get a much greater range of variations. Owing to inaccurate localisation of the tubercle in a certain percentage of the cases, the result justified this assumption. My figures varied from 107 to 230, with an average of 160.

From these figures it seems feasible to say that a certain limitation is imposed on the morphological ear index, and however valuable it may be on morphological grounds, on practical grounds its use is not so great owing to its extreme range of variation.

Helical Index.

This can be rapidly dismissed, as no figures are available for comparison. My range of variation was from 12 to 44, with an average of 24.

Bearing in mind the insufficiency of numbers, I would draw the following tentative conclusions:—

1. That the morphological ear index has certain definite limitations.
2. That the view as to the increase in the size of the ear with advancing age is correct.

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PRELIMINARY COMMUNICATION ON THE INTERRELATIONSHIP OF SOME TRUNK MEASUREMENTS, AND RELATION TO STATURE.

By RUPERT M. DOWNES, M.D., M.S. (Melb.).

1. Nature of Investigation.

THE points to which attention was given in this research were the measurements of the diameters between the cranial borders of the manubrium sterni and the symphysis pubis; between the anterior superior iliac spines, between the tubercles of the crests of the ilia, and between the femoral trochanters; also the total height of the body, and the correlation of these different measurements.

Observations were also made as to the symmetry of the body to determine in what proportion of cases the transverse diameters on either side from the mid-line to the anterior superior spines were equal.

The question was approached from the point of view of considering the sexual differences and age variations, especially in reference to the obstetrical and the great part of the anatomical teaching that the pelvis is both relatively and actually broader in the female than in the male.

The reasons for which I undertook this investigation were, firstly, to test the statement of Addison that the cranio-caudal diameter from the manubrium sterni to the symphysis pubis bears a very constant relation to the height of the body; this constancy would necessarily be of great importance in regard to Addison's method of division of the thorax and abdomen into its several parts.

Secondly, in Berry's clinical atlas of sectional anatomy, many sections are made at particular levels, caudal to the manubrium, so that it was necessary to see if the cranio-caudal diameter of the trunk was fairly constant; if this were not so the sections depicting the structures found at these stated levels would have but little clinical significance.

Thirdly, in suggesting this work to me, Professor Berry very kindly offered the use of indices prepared by him from forty of Addison's measurements and thirty-six of his own; these are to be incorporated with mine.

The number of cases examined was 201; with the exception of eighteen adults from the dissecting room, the subjects were alive—fifty-one females, all of whom had borne children, and fifty male adults, are included, with 100 children, forty-seven of whom were male and fifty-three female. The ages of the whole number range from a few months to 90 years.

The measurements were recorded with Martin's anthropometer, the required points being first of all marked off on the skin with a flesh pencil. This method was found to be more accurate and preferable to the method of placing the pointers on the bony prominences, in the case of interspinous and manubrium to symphysis diameters. In the case of the anterior superior iliac spines their most medial extremities were taken as the limits for measurement, so that the fact of my figures being lower than those of Addison and Berry may be because the latter measured from the most prominent parts of these spines. In this particular measurement I think it probable that individual observers will always differ somewhat from each other.

The height was taken in all cases with the patient in the recumbent position. In connection with this, the well-known phenomenon was noticed that the longitudinal diameter was greater in the recumbent position than in the erect in those cases where the subject had previously been in bed for some hours at least. No constant difference was noted in the case of those who had lain down after having been in the erect attitude for some time previously.

Cranio-caudal Diameter between the Manubrium Sterni and Symphysis Pubis.

I found this to give an average, obtained by Karl Pearson's formula, of 51·12 cm. in males, and of 48·72 cm. in females.

The extremes were 45 and 57 cm. in males; 40 and 53 cm. in females.

The standard deviation from the mean average was 2·64 cm. in the males, and 3·05 cm. for the females.

Only subjects over the age of 21 were included in this table.

The corresponding averages in Addison and Berry's cases combined were 52·7 cm. in males, and 49·4 in females.

From these standard deviations, which are small, it is seen that the points at which sections were made for the clinical atlas abovementioned will be fairly constant in position in all bodies. However, there will be some appreciable difference in the structures found in transverse sections made at measured distances caudal to the cranial margin of the manubrium sterni between subjects who exhibit the extremes of this measurement, which in females I found to be 13 cm. No difference, however, is probable in the structures found at transverse planes in any fraction of this diameter, such as the trans-pyloric, trans-thoracic, and trans-tubercular planes.

Interspinous Diameter.

This diameter in adults over the age of 21 only (forty-two males and thirty-nine females) was found to give the average of 21·98 cm. for females, and 22·62 cm. for males.

The respective standard deviations were 1·94 and 1·21.

Addison's and Berry's tables combined give this diameter as 23·4 cm. in the female, and 24 cm. in the male.

Intertubercular Diameter.

In the same subjects the measurement was 28·11 cm. in females, and 29·19 cm. in males.

The standard deviation was 1·8 and 1·33 respectively.

These two tables show that the male adult pelvis is actually broader than that of the female. This is contrary to some obstetric teaching.

Relation of Interspinous Diameter to Vertical Trunk Diameter.

This relation is expressed as an index obtained by multiplying the transverse diameter by 100, and dividing by the vertical diameter.

The mean average of this gives the results of an index of 43·56 in males, and of 44·67 in females. The standard deviations were 3·48 and 4·14 respectively.

Addison and Berry's cases give indices of 45·5 in males and 46·5 in females.

From this it is apparent that the interspinous diameter relative to the vertical diameter is greater in females than males.

In children I find, however, that the index is greater in males than in females.

Relation of Intertubercular Diameter to Vertical Diameter of the Trunk.

Indices were calculated here in the same manner, the vertical diameter being the denominator. The results were very similar to those of the last table, the index for males being 56·36; for females 57·88.

In children, again, the males had higher indices than the females.

So, if any conclusions are to be drawn from these results in regard to transverse diameters, it follows that the pelvis is relatively broader in male children than in females; but that after puberty the female pelvis becomes relatively broader than the male, though actually it is less in diameter.

Relation of the Vertical Trunk Measurement to the Total Height.

The figures obtained from this relation show that there is an extraordinary constancy in the latter, as previously noted by Addison. Indices were again calculated, taking the vertical trunk diameter as enumerator and the height as denominator, and multiplying by 100.

The results may be tabulated:—

	Index.	Standard Deviation.
Male children	29·89	1·66
Male adults	30	1·15
Female children	30·53	1·58
Female adults	30·57	1·54

This shows that females of all ages have relatively greater vertical trunk measurements than males, while the trunk measurements relatively increase as development proceeds.

This tends to dispose of the popular belief that many individuals have "longer bodies" than others, associated at the same time with greater strength.

Asymmetry.

It is said to be a common belief among tailors that few individuals are symmetrical.

These investigations seem to prove the truth of this, for I find that in 104 cases one side had a greater diameter than the other—that is, a little over half the cases, or 52 per cent. Further, this asymmetry is more pronounced in females, both adult and puerile, especially the former, than in males.

These figures probably underestimate the frequency of this phenomenon, for no subject was adjudged as presenting asymmetry unless there was at least a difference of ·5 cm. in the two lateral measurements; this was done to minimise error in determination of the mid-line of the body.

In conclusion, I wish to thank sincerely the house surgeons and nursing staffs of the Children's, Women's, and Melbourne Hospitals for the help they have given me in carrying out these measurements.

PRELIMINARY COMMUNICATION ON THE ANATOMICAL RELATIONS OF THE THYMUS, CONSIDERED IN REGARD TO "THYMIC DEATH," WITH BRIEF MENTION OF CASES OF ABNORMALITY; WITH FIVE FIGURES.

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In this paper I propose to dwell briefly upon some of the main points brought forward in a review of the literature on the thymus, and by my own researches.

J. A. Hammar concludes a quinquennial review of recent work on the thymus with the expression that he is convinced that any future progress in the physiology and pathology of this gland will depend upon anatomy, and that our present anatomical knowledge of the thymus is in a very incomplete condition.

Whilst I was resident surgeon to the Children's Hospital, Melbourne, and thus in a position to obtain a supply of material for anatomical research, Dr. W. C. Mackenzie suggested to me that some work on the thymus, from the point of view of its topography and relations, would be of value. As this advice coincided with the above-expressed opinion of Hammar, no apology is needed for offering the results of any anatomical investigation upon the thymus to the medical profession. My own personal interest in the subject was still further stimulated by the observation of death in a child found post-mortem to present the condition known as status lymphaticus, and later of two more cases of the same condition.

From a study of the literature of the subject as given in Merkel and Bonnett's "Ergebnisse," and elsewhere, I find that from a clinical point of view it consists largely of accounts of isolated cases of pathological manifestations apparently related to this gland—one of the few glands whose role in the human economy is largely enshrouded in mystery.

The object of this research is to make exact observations from the standpoint of the "mechanical theory" put forth to explain the causation of "thymic death."

Historical.

A few remarks may be made on the history of the views held on the structure of the gland from early times. The seventeenth and eighteenth century observers classed the thymus as a conglomerate gland, as it had a somewhat similar lobulated appearance to that of the pancreas, and, though none could ever be found, it was thought to have a duct running to the trachea, œsophagus, or mouth; therein was a difference from the type of conglomerate gland. Bellinger (1717), however, described a duct running from the thymus to end with the salivary duct in the mouth; but he was little supported in the discovery. What we now know as lobules were spoken of as cells, cysts, acini, folliculi, globuli, or frustuli.

For two centuries the question of the existence of cavities in the lobes of the thymus was a great morphological problem, and in connection with this the question of dependence or interdependence of the lobes, and the latter remains as a debated point even to the present day.

The original views that the cavities occurred spontaneously on section by evacuation of the fluid contents were greatly modified in the course of time. For it was soon found that the cavities were not always apparent, and as this was supposed to be dependent on the fact that the walls were adherent, means were sought to demonstrate the cavities. Such methods were used as maceration in water, or injections of air, water, mercury, or molten wax into the organ. The formation of the resulting cavities was portrayed by Hugo and Moraud as usually consisting of one discrete cavity in each lobe, with uneven and lacerated walls traversed by red flesh-like strands, containing fluid, and large enough to admit the tip of the thumb, with the inner wall also presenting the openings from the cavities of smaller lobules. The teaching was now generally one of cavities with intercommunicating ducts; however, there were opponents to this view, for Bassius (1771) threw doubt on the existence of the cavity described by Bartholius and Haller, but finally came round to the generally accepted view.

In 1811 a reaction against the view of cavity-formation set in, mainly owing to the work of Lucae; he came to the conclusion that the cavities described by older observers were artificially produced.

Anatomy.

The gland is most frequently described as consisting of two lobes, but other divisions have been described by different writers—as Bartholinus, who spoke of three lobes, Becker of four to six, and Cruchet of four.

Hammar considers that all the cases of lobes other than two in number are abnormal, while Piersol states that the variations are due only to the irregularities of the investing fibrous tissues.

In five of my cases the gland appeared to be formed of a single body until close dissection disclosed the fact that the two halves of the gland were entirely distinct, with no central connection, as has been sometimes described. One of the cases apparently consisted of five lobes, another of four, but the increased number was due to the two lateral lobes being subdivided into further potential lobes, which were continuous with each other by glandular tissue. I have not yet seen a case where the gland was not essentially divided into two lateral lobes.

The gland is situated partly in the thorax and partly in the cervical region, the proportions varying in individual cases. Sometimes one or both lobes rise to the level of the glandula thyreoidea, more usually falling short of it. Caudally the gland reaches to about the level of the cranial margin of the fourth chondrosternal articulation, though variation is again present here. In some cases it is said to reach to the diaphragm, though this is denied by several writers, who say that such an extension is only by means of fibrous tissue. A connection is frequently found between one or both cranial cornua and the corresponding lobes of the thyroid gland, consisting in some cases of glandular tissue, in others of fibrous tissue only; this strand has been called the ligamentum suspensorium by Piersol, and seems to be the remains of the part of the gland here present in embryonic life in its passage from the third branchial cleft, from which it is developed, to its final more caudal situation.

Relations.

The relations of the gland are very constant, and subject only to slight variations, the dorsal being most worthy of consideration.

Ventrally we find, after removal of the superficial structures, the sternum, costal cartilages, and Mm. intercostales interni, articulatio sterno-clavicularis, clavicle, Mm. sterno-cleido-mastoidei, sterno-hyoidei, and sterno-thyroidei, with an uncovered area between the two last muscles; internal mammary vessels, retro-sternal and præ-thymic lymphatic nodes, with some of the thymic vessels in the connective tissue of the anterior mediastinum.

In case No. 6 of my series the V. anonyma sinistra was found to run ventral to both superior cornua of the gland.

In connection with the surgery of the gland, the fact that this abnormality may take place is of considerable importance; for in complete or partial thymusectomy, without previous removal of part of the manubrium sterni—the method that has been more generally followed in the few cases reported of operation on the gland—this vein would be in great danger of being wounded.

Farret, quoted by Kaplan, has seen cases in which the left cornu lies ventral to this vein, but apart from his observation I have found no further reference to this unusual relation.

Laterally lie the pleuræ and lungs in the thorax, the glandular margins intervening between the pericardium and the lungs, sometimes coming into contact with the roots of the latter. On the right side the A. carotis communis, N. phrenicus, and dorsally as well the N. vagus, with its recurrent branch; in the neck, the Aa. anonyma and carotis communis. On the left side the A. carotis communis, N. phrenicus, and dorsally as well the N. vagus.

Dorsally the relations are more numerous, being the pericardium, separating the gland from the atriæ and their auricula, and part of the bases of the ventriculi; Aa. pulmonalis et pulmonalis sinistra, aorta ascendens and arcus, V. cava superior, Vv. anonymæ and thyroidea inferior, Aa. anonyma, thyroidea ima if present, carotis communis sinistra, and sometimes subclavia sinistra; Nn. vagi—laterally, too—and laryngei inferiores, cardiac rami from the Nn. vagi and sympathetici. The last I have only found mentioned by Rieffel and Lee Mée, who state that the N. cardiacus cervicalis inferior dexter is directly in contact with the gland, and certainly has some influence in the production of syncope of thymic origin. In addition, the ventral surface of the trachea, with its sides also in some cases, for Rieffel and Le Mée found the gland stretching into the tracheo-carotid space in eleven cases out of thirty-four, and there coming into contact with the œsophagus and Nn. laryngei inferiores. Finally, we find the retro-thymic lymphatic nodes and vessels of the thymus.

The dorsal relations are those of most importance in considering the possibility of mechanical factors, being etiological in the production of "thymic death."

Many cases have been reported in which, after death in children, some of whom had presented the condition of stridor previously, the thymus has been found to be abnormally enlarged, with compression of the trachea. Such compression has also been noted in a tracheoscopic examination. It does not seem likely, however, that this tracheal narrowing is the essential factor in the production of sudden death.

No very marked symptoms have been noticed and recorded as due to arterial interference, but some cerebral anæmia is a possible result.

In a few cases definite signs have appeared of venous engorgement; but, though it has been propounded as a cause of death, vascular obstruction does not seem a sufficient cause, so that the nerves alone remain to be considered.

Those that have been blamed in this connection are the Nn. laryngei inferiores, phrenici, vagi, and sometimes some of the cardiac rami. The Nn. laryngei inferiores have been named because it is considered that stridor may be a result of their involvement, and because in some part of their course they usually lie directly dorsal to the thymus. I have found that this relation is inclined to be more intimate on the left side, the nerve being in actual contact with the gland in my case of status lymphaticus. But though I believe that in this way the stridor may be caused, yet many children have died without any stridor, so that as a rule, to which there may, however, be exceptions, it does not seem likely that involvement of these nerves is the important factor. Exceptions are, perhaps, found in those cases which die during a paroxysmal suffocative attack, accompanied by stridor; but even in some of these no post-mortem signs of suffocation are found, and tracheotomy has been of no avail. This would not be the case if spasm of the glottis were the whole trouble.

The Nn. phrenici have frequently been considered to have the chief influence in this result. The right nerve has been repeatedly described as bearing the more intimate relation to the gland. Cruchet and Rieffel and Le Mée think the opposite to be the case, the left nerve sometimes grooving the gland, while the right passes more rapidly dorsalwards. I do not find much difference in this relative relation, though the left nerve was in actual contact with the gland in five of my cases, the right nerve in two cases. The left nerve was nearer the gland more frequently than the right, and in all it was parallel to the margin of the gland through a greater distance. Here also the mode of death does not seem compatible with phrenic paresis.

The Nn. vagi also have been indicted. They do not approach so closely to the gland in most cases as do the Nn. phrenici, but still are susceptible to adverse pressure from it. This pressure might be felt by the fibres of the thoracic cardiac rami before they are given off. I have found that the right nerve tends to be more intimately related to the gland than the left.

In regard to the cardiac nerves, which do not seem to have attracted much attention, Rieffel and Le Mée place importance on the R. cardiacus cervicalis inferior in the causation of syncope, while Kaplan states that the vagal and sympathetic branches of these plexus may be compressed.

In all of my cases where such vagal branches were found they passed dorsal to the gland in comparative proximity, and in suitable positions so as to be dorsally compressed by an enlarged gland. The branches on the right side were more intimately related to the gland, being in contact in two cases, though they did not always follow the text-book description of their courses.

It is obvious from what has gone before that all these nerves, vessels, and organs will be to some extent affected by an increased pressure due to an enlarged thymus, and it is only a matter of secondary importance as to which of the structures compressed is most concerned in the production of the fatal results.

To me it seems most probable that the branches to the cardiac plexus are those in which interference with their functions would have the greatest effect in causing sudden death. In regard to this it is to be remembered that cardiac failure has been noted to precede respiratory failure in some cases at least, including one of my own. The question, however, is largely physiological, my chief purpose having been to consider the question from an anatomical standpoint as to whether the various vital structures are in a position to be compressed by an enlarged thymus gland. I think that they are certainly compressed, and so take at least a share in causing death.

Involution.

The question of the involution of the thymus is responsible for much difference of opinion. One group of observers considers that the gland has its maximum development at the time of birth or up to the end of the second year, degenerating after this, but present till puberty, when it disappears. Later researches, however, seem to show that the gland has its greatest weight at puberty, while it is present in part throughout the whole of life.

The normal weight of the gland is also very greatly disputed. One class of writers gives varying weights up to 10 gms., while another places it as anything between 10 and 35 gms. The reason of this discrepancy is that the thymus displays a tremendous variation in its size in different pathological conditions, so that it is difficult to determine what is the normal weight of the gland. In diseases of infants, associated with wasting, the gland has a very low weight, while in that of status lymphaticus or lymphatism the weight is very much greater than normally.

Personally, I should agree with those who consider the normal weight to be under 10 gms.

Socia Thymi Cervicalis.

Previously, as far as I can ascertain from the literature, only three cases of this condition in full-term children have been found, with a fourth one in a fœtus. Harman described the first in a full-term fœtus, while Gertrud Bien's cases were in new-born children. I have found an example of this abnormality in an infant aged 6 months, who presented the condition of status lymphaticus.

The glandular structure was situated in the right carotid triangle, extending from the level of the body of the os hyoideus to that of the body of the cricoid cartilage. It was pyramidal in shape, crossed laterally by the N. descendens hypoglossi, N. vagus, and V. jugularis interna; medially it was related to the A. carotis communis and its terminal branches. Its cranial base was closely related to the N. hypoglossus, the M. digastricus, and the submaxillary gland. In close relation to it also was an accessory salivary gland.

A direct connection with the main gland was present by means of a thin suspensory ligament, which was glandular in structure.

In Harman's case the main thymus was an undivided mass, with a cranial prolongation on the left side of varying size, consisting of thymus substance throughout, which reached slightly cranial to the ala of the thyroid cartilage. Superficially the socia was covered by the V. jugularis interna with the Aa. carotides and N. vagus as deep relations, the Aa. carotides bearing the same relation to the gland as in my case. A neck of the socia just caudal to its extremity was encircled by two divisions of the N. vagus, forming a loop, from the ventral of which the N. descendens cervicis arose and crossed the socia superficially.

In my case no such loop was present, the N. vagus lying lateral to the gland, while the N. descendens cervicis arose normally and crossed the lateral surface of the socia.

The arterial supply was from the Aa. carotis communis, and externa and thyroidea superior; vein to the V. thyroidea superior.

In each of Bien's cases the prolongation from the left lobe reached to the hyoid bone, as in my case, which was, however, on the right side.

In one case the lobe presented a branching Y form at its cranial extremity, the whole cervical prolongation lying superficial to the V. jugularis interna and Aa. carotides; the dorsal fork of the bilobed extremity was enlooped by a branch from the N. vagus joining the R. descendens hypoglossi, running with it for a short course over the ventral surface of the gland, and then rejoining the N. vagus.

In the second case a flattened most cranial lobule was separate from the remaining cervical prolongation, all of which was superficial to the Aa. carotides and V. jugularis interna. The N. vagus lay medial to the gland, while the R. descendens hypoglossi embraced its ventral border laterally.

I shall now show lantern views of a series of five transverse frozen sections of a full-term stillborn female child.

Section I.—This section is made at a point ·9 cm. cranial to the manubrium sterni. It shows the cornua of the thymus in section close to their cranial extremities, the left one being a little larger than the right.

The right lobe is in contact with the A. carotis communis and the trachea, with the V. jugularis interna somewhat dorsal, but chiefly lateral, to it. The N. phrenicus dexter lies dorsal to the V. jugularis interna on the ventral surface of the M. scalenus anterior.

The N. vagus dexter is in the dorsal angle formed by the A. carotis communis and the V. jugularis interna, separated by the former from the thymus. The left lobe presents very similar dorsal relations, but in addition the V. thyroidea inferior is in contact with its medial side, while the œsophagus lies dorsally; in this case the œsophagus is rather to the left side of the trachea.

The N. vagus is rather nearer to the left than the right cornu.

Ventrally, both cornua are in contact with their respective Mm. sterno-thyroidei et sterno-hyoidei, with the Mm. sterno-cleido-mastoidei superficial thereto. Small portions of the most cranial parts of the clavicles appear in this section, and the heads of the humeri in their glenoid fossæ; the section has passed more caudally on the left than on the right side.

Section II is made at a point ·15 cm. caudal to the cranial margin of the manubrium sterni.

The thymus appears indistinctly divided into two lobes now lying in contact with the dorsal surface of the manubrium sterni and the intervening depressor muscles of the larynx.

Portion of the first rib is seen on either side embracing the apices of the lungs and their costal and parietal pleuræ.

The right margin of the gland is in relation to the V. anonyma dextra, and dorsally in immediate contact appears the V. anonyma sinistra.

The A. anonyma is separated from the gland by this last vein only, and in its turn lies directly ventral to the trachea. The œsophagus is again apparent, somewhat to the left of the trachea, and the A. carotis communis sinistra is separated from the gland by the V. anonyma sinistra. Dorsal to the leftmost portion of this vein is the A. subclavia sinistra in contact with the medial surface of the lung.

The V. axillaris is seen lateral to the first rib on either side, accompanied by its artery.

Portion of the second rib is also seen on either side in its dorsal part.

The N. phrenicus dexter is seen in close relation to the right surface of the V. cava superior, while the left one lies dorsal to the leftmost margin of the V. anonyma sinistra. The N. vagus sinister appeaars slightly dorsal and medial to this, lateral to the A. carotis communis, so that the two latter nerves are separated from the thymus only by the V. anonyma sinistra.

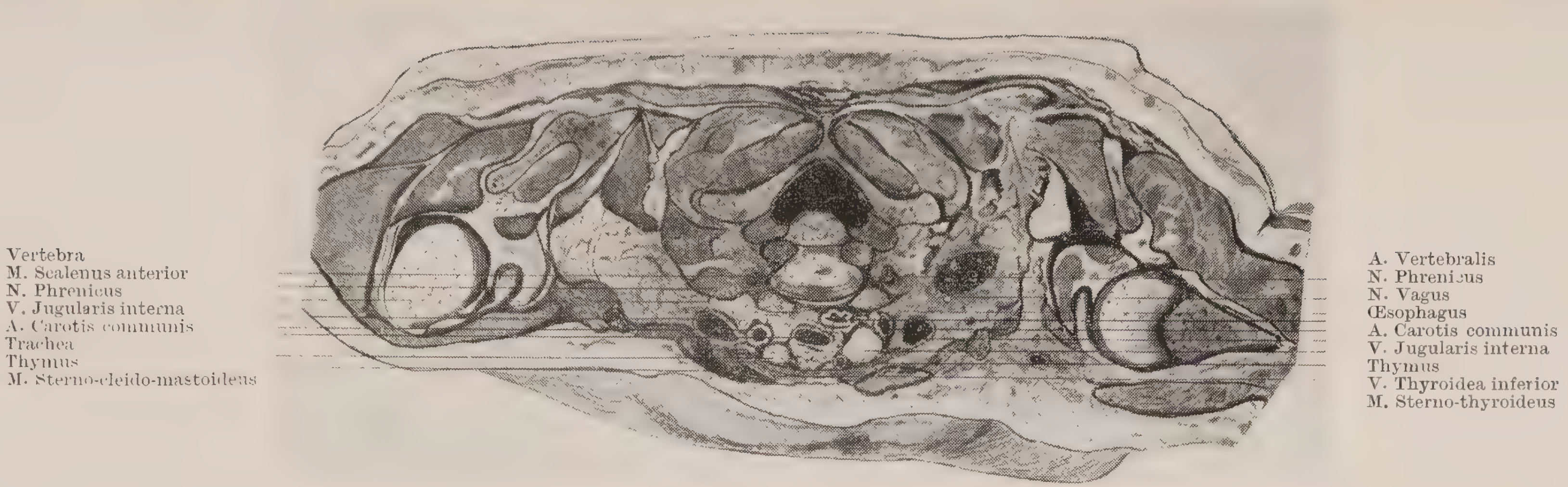
The N. vagus dexter appears lateral and dorsal to the trachea as a flattened band, due to the fact that the Rr. cardiacus thoracales are just being given off at this point. It is here separated from the thymus only by fat and connective tissue.

The thoracic duct is seen dorsal to the A. carotis communis sinistra, and lateral to the œsophagus.

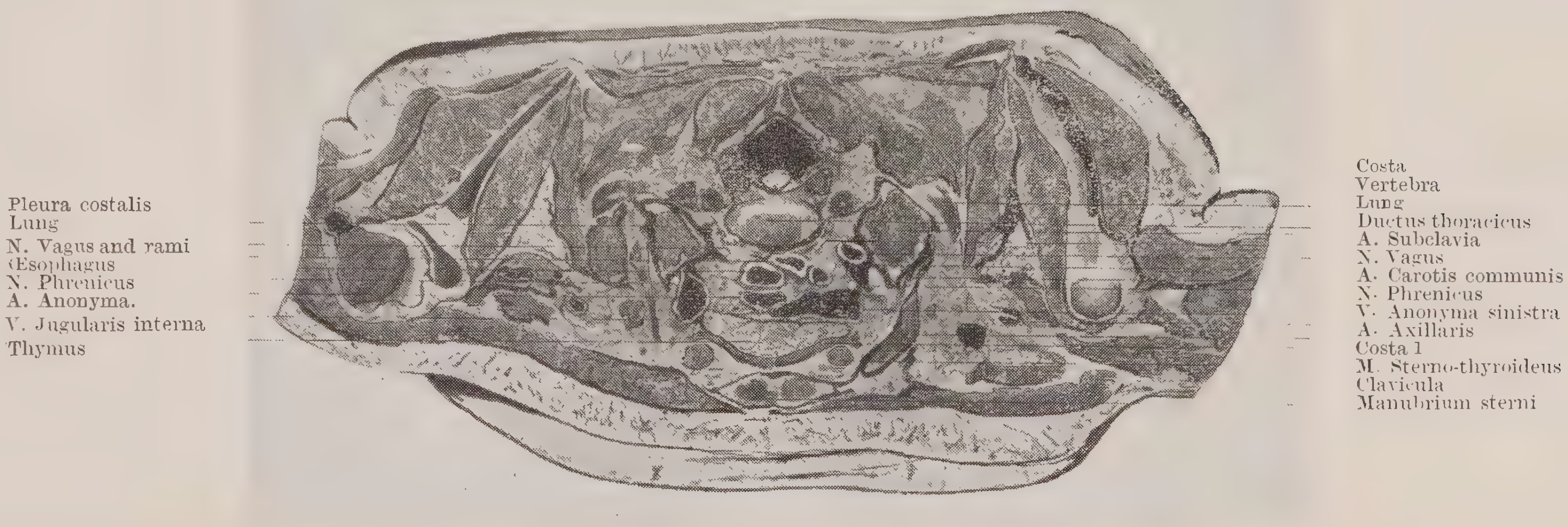
Section III is cut at a point 1·24 cm. caudal to the cranial margin of the manubrium sterni.

The thymus is here seen to occupy a large proportion of the thoracic cavity, and to show its irregular divisions into two lobes, the left one occupying a greater area than the right.

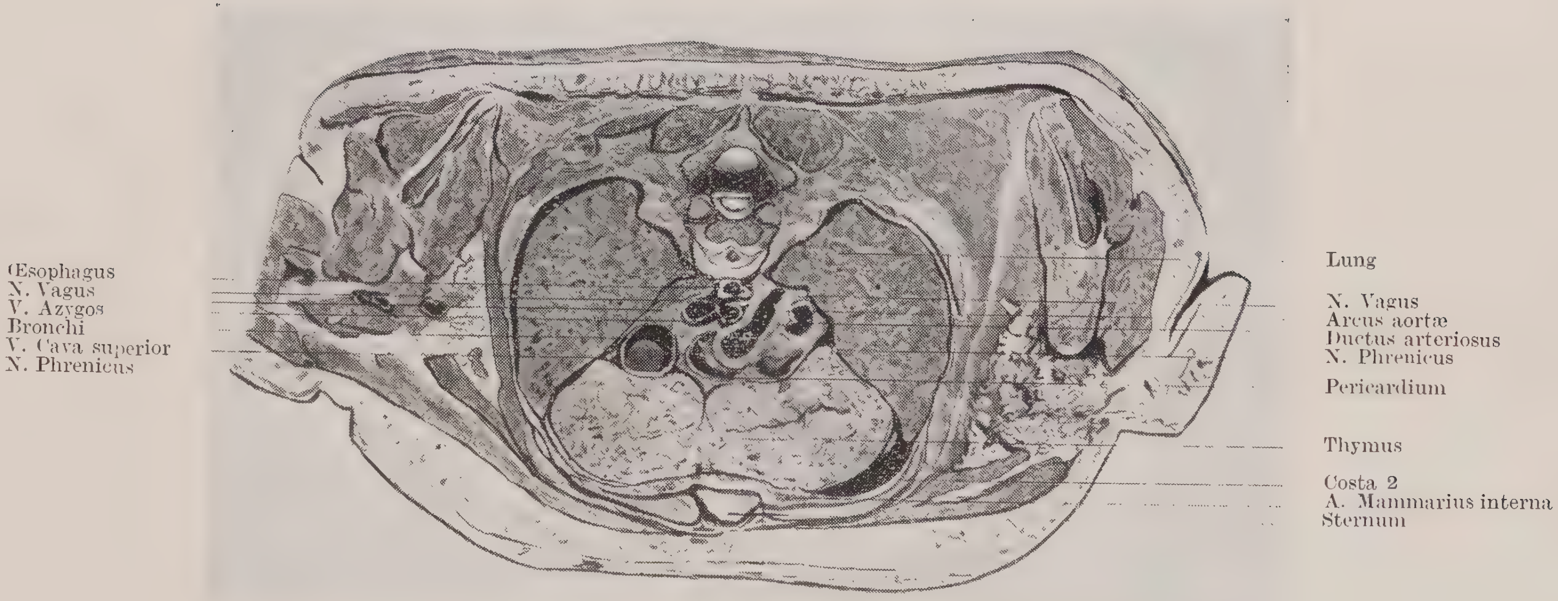
Dorsally it is now seen to be in direct relation with the vena cava lying beyond the limits of the pericardium, the fibrous layer of the pericardium, and an intervening space between the latter and the arcus aortæ and ductus arteriosus. Still further dorsally the trachea has just divided into the two bronchi, the œsophagus now lying in the mid-line dorsal to the left bronchus.



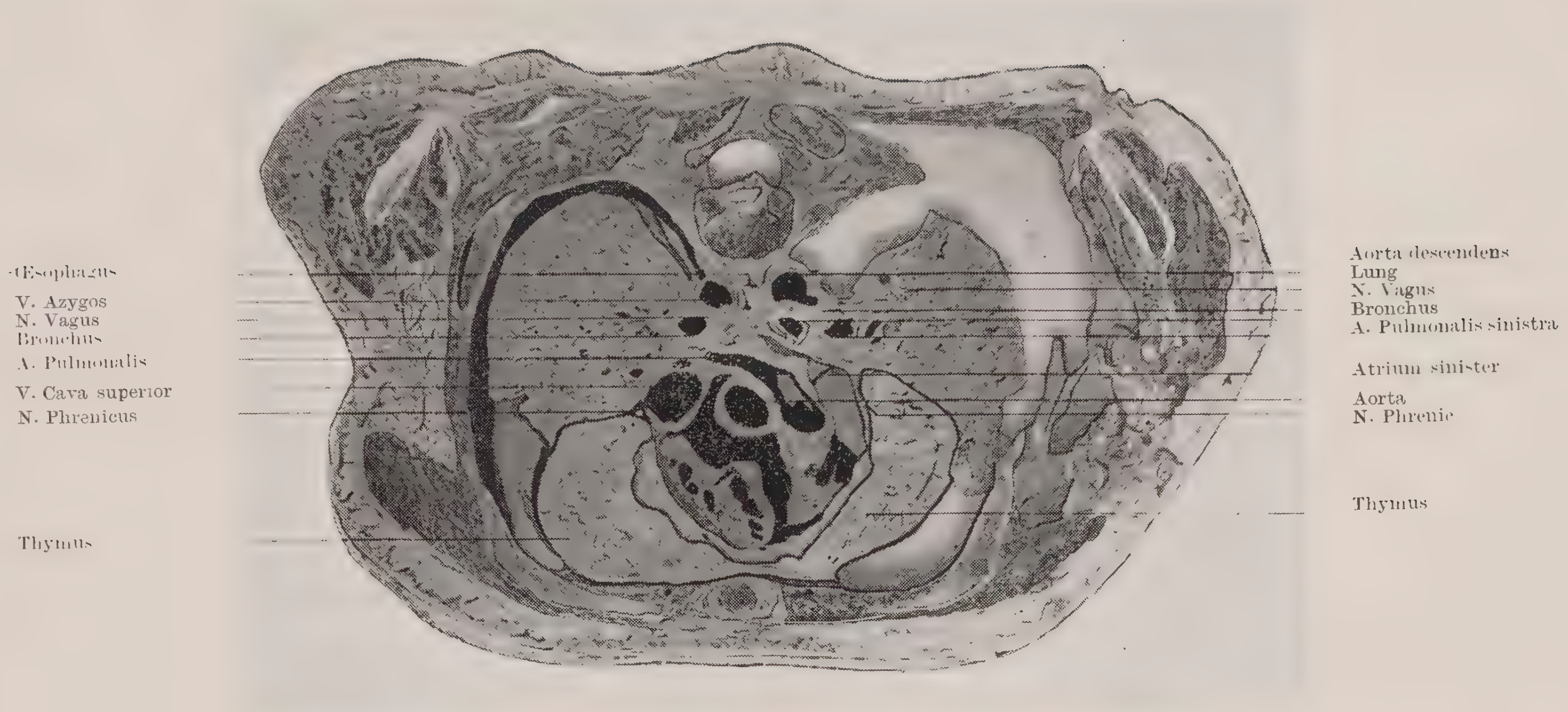
Section I.—·9 cm. Cranial to the Manubrium Sterni.



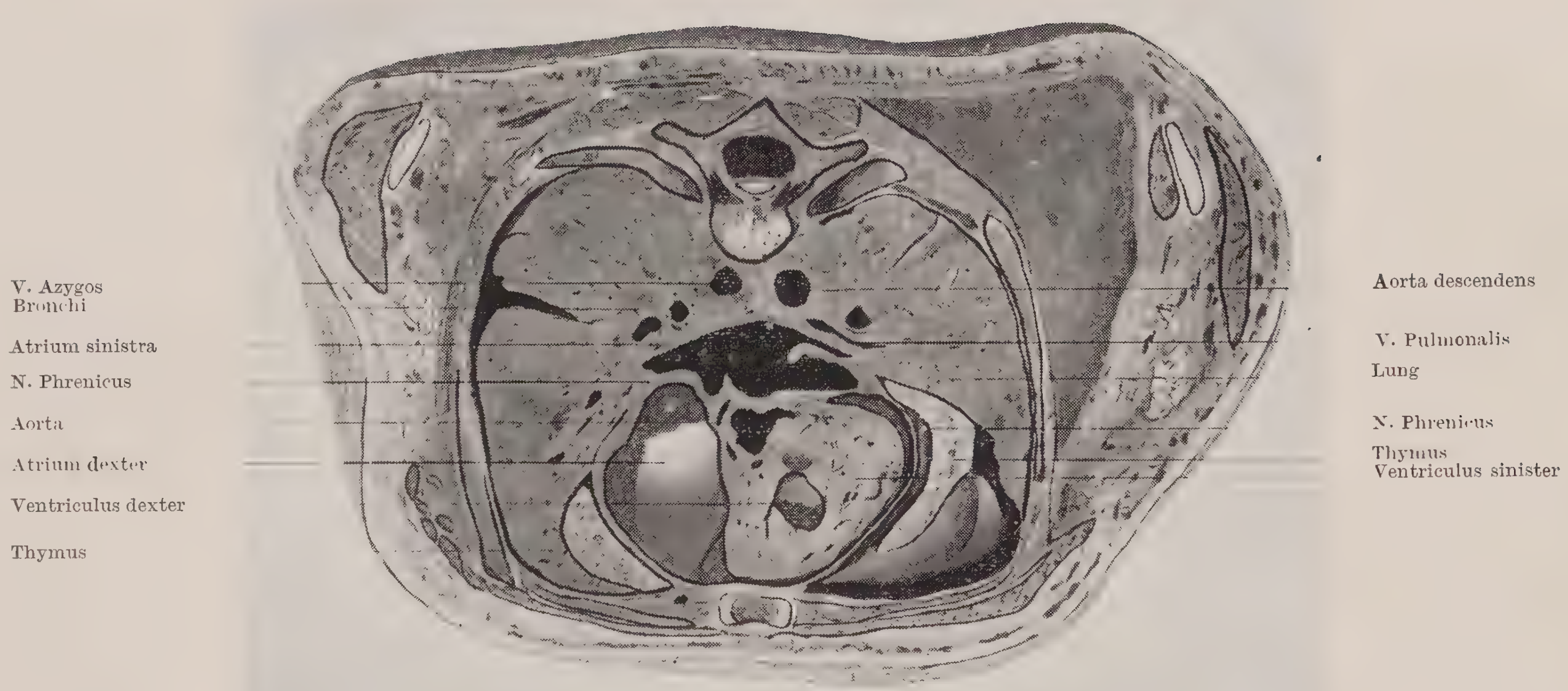
Section II.—·15 cm. Caudal to Manubrium Sterni.



Section III.—1·24 cm. Caudal to Manubrium Sterni.



Section IV.—2·12 cm. Caudal to Manubrium Sterni.



Section V.—2·82 cm. Caudal to Manubrium Sterni.

At the lateral margins of the dorsal surface the Nn. phrenici are apparent, on the left side the companion artery being also apparent. It is seen distinctly in the actual specimen that these nerves are situated outside the fibrous capsule of the gland, closely bound down to it by the pleura. In this case there is, therefore, no difference in the intimacy of relation of these two nerves to the thymus.

The Nn. vagi are seen lying some distance dorsally, the right nerve appearing just to the right side of the œsophagus, and separated from the gland by the right bronchus, the right margin of the aorta ascendens and the pericardium. The N. vagus sinister appears in the space between the ductus arteriosus, left margin of the arcus aortæ, and mediastinal surface of the left lung, and is here separated from the gland by the ductus arteriosus (caudal to this level by the A. pulmonalis sinistra) and pericardial cavity.

Ventrally the sternum and second ribs form relations with the internal mammary vessels.

Laterally the lungs on either side surround an appreciable part of the gland.

The pleura is closely adherent to the surface of the gland on its ventral, lateral, and part of the dorsal surface, binding the Nn. phrenici to it, its ventral line of reflection being situated some distance lateral to the margins of the sternum.

Section IV is at a point 2·12 cm. caudal to the cranial margin of the manubrium sterni.

Part of the section was broken in preparation.

The thymus is now becoming diminished in size, the right lobe being more marked, the whole gland surrounding the pericardium and heart on the ventral and lateral surfaces, and in turn being embraced by the lungs laterally, by the corpus sterni and the third ribs ventrally. There are seen also in the section the auricula sinistra, sections of the A. pulmonis communis, aorta, V. cava superior, and atrium dexter.

The N. phrenicus sinister lies now medial to the gland between it and the pericardium; the N. phrenicus dexter retains the same relative position as in the last section.

The N. vagus sinister is seen at the vntral portion of the left lateral margin of the aorta, the N. vagus dexter on the ventral surface of the vena azygos dorsal to the bronchus.

Section V is made at a point 2·82 cm. caudal to the cranial margin of the sternum, and the extremities of the two lobes have been almost reached, the left lobe being longer in a ventro-dorsal direction, but being more narrow in a transverse direction. The left lobe impinges on the ventricles of the heart, with the pericardium and N. phrenicus intervening. The right lobe is in relation to the atrium dexter, but the N. phrenicus is some distance dorsal to this to the right side of the inter-auricular septum.

In conclusion, I wish to express my most cordial thanks for much help extended to me in this research by Dr. J. H. Anderson, Professor Berry, Dr. Zwar, and Mr. W. Preston.

V.

SECTION OF PATHOLOGY.

OPENING REMARKS.

By the President, WILTON W. LOVE, M.B., C.M. (Edin.).

IT is not my intention to inflict anything in the nature of a Presidential Address upon you for the dual reason that the time at the disposal of our Section is already too short to allow of the papers contributed being read and discussed, and that there is little that I could say which could be of sufficient importance to justify my taking up your time with it. I am deeply sensible of the honour conferred upon my State by the selection of one of its members to occupy the Chair of this Section, and also of my own unworthiness of the honour. In a State like Queensland, where, until this year, there has been no University, and where no medical school exists, the subject of pathology cannot necessarily receive that amount of attention it deserves; but the importance of the subject is becoming so insistently assertive in the daily work of the surgeon and the physician that one must perforce endeavour to keep somewhat abreast of the swelling tide of pathology. In my student days—now alas, a quarter of a century past—pathology consisted largely of morbid histology, a very little chemical pathology, and some parasitology. Since then, the advent and growth of bacteriology has revolutionised our ideas of pathology; bio-chemistry has developed to an unforeseen degree; parasitology has become a special science; clinical pathology, and hæmatology with its Widal, its Wassermann and its precipitin reactions have become necessary adjuncts of the physician's armamentarium; while the pathology of the living, so ably described by Moynihan, has become an equal necessity to the surgeon. On every side the march of medicine and surgery has been *pari passu* with that of pathology; and it seems to me that it is from the domain of bio-chemistry that further laurels for the crown of medicine will in the near future mostly come.

For the first time this Section has had incorporated with it the subject of "Tropical Medicine"—a subject which, thanks to the foresight of Mr. Chamberlain, has made great headway during the past decade—and it is high time that a country, one-third of which lies within the tropics, should pay special attention to it. We have with us to-day the Director of the Australian Institution of Tropical Medicine, Dr. Anton Breinl, a man who is pre-eminently fitted for the position he holds, and to whom we look with confidence for important additions to our knowledge of the ætiology and treatment of the diseases of tropical Australia. The Tropical Institute is situated at Townsville, and is yet in its infancy—under the joint control of the Federal Government, the Australian Universities, and the Queensland Government. We hope before long to see a degree in tropical medicine conferred by these Universities after post-graduate study at the Institute, and the establishment of research scholarships. All this, however, cannot be done without the sinews of war, and I think we can strengthen Dr. Breinl's hands by asking Congress to impress upon the Federal and State Governments and the Universities the importance of the undertaking and the necessity for providing additional money for research scholarships, and thorough equipment and maintenance. Dr. Breinl will have the opportunity of showing "the mettle of his pasture" to-morrow morning in his paper on the "Objects and Scope of Tropical Medicine," and in opening the discussion on ankylostomiasis, and I would ask a good attendance of members to hear his papers and see his specimens.

AN INQUIRY AS TO THE INCIDENCE AND CAUSE OF GOITRE IN TASMANIA.

By J. S. Purdy, M.D., C.M. (Aberd.), D.P.H. (Camb.), F.R.S.. E.
Chief Health Officer, Tasmania.

Previous to leaving New Zealand early in 1910, I was much interested in a circular forwarded the previous year by Dr. Colquhoun, of Dunedin, to all medical practitioners in the Dominion, asking the following questions:—

1. Is goitre endemic in your district, or sporadic?
2. Is there any permanent cause in your district which may lead to goitre, such as glacier water, water otherwise charged with foreign matter, or are there any other circumstances which, in your opinion, may be etiological?
3. Is exophthalmic goitre common in your practice? Can you give any personal experience which may throw light on its etiology?
4. Can you make any suggestion as to the treatment of either form?

On arrival in Tasmania, I made inquiry from Dr. Gertrude Halley, Medical Inspector of Schools, as to the incidence of goitre in that State. Dr. Halley furnished to Dr. Colquhoun the following note, which was embodied in his excellent paper on "Goitre," given at the annual meeting of the New Zealand branch of the B.M.A., Wellington, on 22nd February, 1910:—"Goitre is prevalent in some parts of Tasmania, especially in the Huon district. At one school, Geeveston, 46 of the children out of 111 examined were found to be suffering; some in the infant classes. In another school there were 4 children attending school from one family. On inquiry, I found that 9 out of 11 in that family were affected. The water is, as far as I could gather, all rain-water. In Geeveston, the people are all closely related; the parents of many were affected, and, I believe, the grandmother (old Mrs. G.) of very many of the children. In no case among the children was the goitre interfering with their educational progress."

A circular repeating the queries of Dr. Colquhoun elicited fifty-four replies from medical practitioners in Tasmania. Twenty-one reported goitre to be endemic in their districts, which were Port Esperance, Franklin, and Geeveston, all towns in the Huon; Hobart, New Norfolk, Ellendale, Scaggy Flat, Bothwell, Oatlands, Longford, Deloraine, Sheffield, and Queenstown.

The Huon.—Dr. Gordon Allport, Port Esperance, writes:—"I should say endemic. The water consumed is mainly rain-water. I do not know of any other etiological factor."

Dr. Murray Anderson, Franklin, The Huon, writes:—"Markedly endemic; a large proportion of the people have it, especially in the valleys and parts most removed from the sea coast. Drinking-water theories are largely negatived as a cause in my district. A large proportion of cases known to me drink exclusively rain-water. This is a very windless district, and a stagnant damp atmosphere its most salient characteristic."

Dr. Cummings, Franklin, The Huon:—"Endemic. Goitre seems to be more prevalent in the highlands and gullies, which would seem to point to glacier water; but, from inquiries made, most of the people drink tank-water."

Dr. Stephens, Geeveston, The Huon:—"Endemic; very prevalent, I should estimate that about 10 per cent. of the people of this district are affected more or less with goitre. Most of the water drunk is rain-water off shingles or iron roofs. There is practically no limestone in this district, which may possibly have some bearing on the undue prevalence."

Hobart.—Drs. Butler, Giblin, Hardcastle, and Sprott report that goitre is endemic. Dr. Giblin:—"Simple parenchymatous goitre very common; quite 10 per cent. of young adults."

Dr. Ireland:—"Endemic. The water from Hobart comes from springs on the mountain fed by snow-water during half the year. It is considered an excellent water, but is said to be deficient in lime. The dwellings in the city are badly ventilated, the soil is charged with septic matter, and dust-storms are frequent."

New Norfolk.—Dr. Allwork:—"Endemic. Watercourses highly sedimentous, and in winter largely supplemented by melted snow. Most supplies more or less peaty."

Hamilton.—Dr. Morgan:—"Endemic in two localities, Ellendale and Scaggy Flat. Both localities are low-lying and very damp, both have a large rainfall, and both are at the foot of high hills."

Bothwell.—Dr. Gairdner:—"I should say endemic. It has occurred to my mind: Is it attributable to the storage of drinking water in galvanized tanks?"

Oatlands.—Dr. Jones:—"Endemic. All the goitre cases occur in those living on elevations of 1,400 feet or over, and who use well or creek water."

Launceston.—Dr. Pardey:—"Goitre is frequently met with in Launceston, but mostly in anæmic girls, and disappears generally after treatment for anæmia, and varies in size according to the general health. I seldom see a goitre in a male, so that it must in these cases be due to something connected with the female generative system."

Dr. Pike, Launceston :—" Endemic and very common. I believe it is due to the water supply."

Dr. Webster, Launceston :—" Endemic. For the last nine years I have been wondering as to its cause. In winter and spring there is, of course, a good deal of melted snow-water in the St. Patrick's River, and found on the slopes of Mount Barron and Mount Arthur. I believe there is a comparative absence of iron in the water. Near river levels in Launceston, I consider the air generally heavily charged with moisture and, therefore, relaxing."

Longford.—Dr. Mason :—" I have worked in this district for forty-two years, and goitre has been endemic all along the foot of the Western Tiers during that period. In the first ten years of my practice, I became very well acquainted with goitrous cases of all sorts in Herefordshire and Derbyshire. Until better advised, I retain the old theory that goitre is caused by excess in the mountainous waters of calcareous and magnesiate salts."

Deloraine.—" Dr. Cole :—" I believe it to be endemic. The country adjacent to the western tier of mountains is where the disease is most prevalent. For a great part of winter snow lies on these mountains, and the water of the creeks is fed by the snow. The country also is mostly of a limestone character."

Queenstown.—Dr. Love :—" Endemic, both in the simple and the exophthalmic form. All the drinking water round Queenstown is charged heavily with various minerals. The rain-water contains, I am told, an appreciable quantity of H_2SO_3. Surface water contains lime and various sulphates and much organic matter. Sunlight is seldom seen here. Rain is almost continuous. Houses invariably damp. Moulds and fungi common.

Gormanston.—Dr. Walpole :—" I only know of one medium-sized goitre (in a woman) in the district at present. There are a few cases of fulness of the thyroid. One would hardly class them as goitre. One girl died suddenly two or three years ago. ' Scabbard trachea,' due to goitre, was found by me at the post mortem.

" I saw one marked case in Strahan in a woman thirteen years ago; also one in a woman at Queenstown, who was on a visit from Hobart; this was some years ago. As to exophthalmic goitre being common, I should say yes. One per cent. of the women are affected. One man some little time ago died suddenly, and I saw another case in a man the other day. Absence of lime in drinking water is an idea I have seen suggested elsewhere. There may be something in it."

Sheffield.—Dr. Ratten :—" Endemic. My last forty cases of simple goitre seem to be caused by drinking water from springs or underground wells (common in this district). This water is hard. Out of those cases of which I have kept slight record, all have been entirely or partially caused by drinking this water. All but one of these cases were females from 16 years upwards. With regard to exophthalmic goitre in Tasmania—except in Hobart, Sheffield, and Queenstown—the concensus of medical opinion appears to be that it is only of rare occurrence."

With regard to Dr. Colquhoun's inquiry in New Zealand, it is shown that simple or passive goitre is more prevalent in certain areas than elsewhere in the Dominion. Thus it is very evident from the report of Dr. Sutherland, of Lawrence, with regard to the Rangahera district, situated on the Molyneaux River, at the foot of the Blue Mountains, that goitre is endemic there; in fact, "it is the exception to find a young woman (18 to 25) in the district without simple goitre. The Rangahera Valley is very narrow, and the hills rise on each side of it to a considerable height. A mist usually lies along the valley at nights. The heat of the sun at midday is strong, but it is not of long duration, especially in the winter time. Water used in mostly from springs or small hill streams. Sun's rays are strong when present, but only for short periods of each day. Bush and scrub cover most of hill sides."

Professor Marshall, of Otago University, after reviewing the geological structure of the localities where goitre was endemic in New Zealand, writing to Dr. Colquhoun, states: "In looking through this list, you will probably be struck by the general occurrence of Tertiary deposits and Jurassic sediments in the districts you mention. I do not think that any importance is to be attached to this, as these are the rocks in most of the settled parts of the Dominion. I can see no one character of physiography or structure markedly, or even generally common, to the districts to which you refer."

Dr. Colquhoun states that the districts enumerated include places near the sea level, valleys, highlands, open country like the Canterbury Plains, and bush lands as in the Urewera country, and are marked by great variety of rainfall and climate.

After discussing the theory of Professor Farr, of Canterbury College, that goitre is probably due to radio-active substances present in water, Dr. Colquhoun, to sum up, states: "I think we are justified in making the following statements concerning passive goitre in New Zealand:—

"1. That the female sex about the age of puberty, and for some years afterwards, is specially predisposed to enlargements of the thyroid gland.

"2. That the existing agency is to be looked for in the drinking water.

"3. That we must look for something which is common to rain-water, artesian wells, and the natural hot springs.

"4. That the only common factors in these waters are the radio-active substances or their derivatives, but that the physical evidence is not as yet sufficient or conclusive on that point."

With regard to the association of goitre in parts of the North Island of New Zealand with waters from the hot springs, I would point out that seldom are the waters from the hot springs drunk, certainly not at Whakarewarewa, although, as Dr. Te Rangihiroa points out, the food is cooked there in the natural steam holes.

I am inclined to think that the Tasmanian evidence, which shows that goitre is prevalent where the people either drink glacier water or live on flats through which glacier water drains, and which must form part of the subsoil water, points to the enlargement of the thyroid being probably due either to some organism which flourishes in such districts or to some property of such waters. As Dr. Colquhoun pointed out, " Water has been universally suspected, and apparently with reason, as the chief factor in producing goitre."

It is said that in Derbyshire, people who boil the water do not become goitrous. Dr. McCarrison, of Gilgat, claims that by boiling the drinking water it is rendered non-goitrogenic, and that after filtering the natural water he can produce goitre at will by administering the filtrate to men and animals. He also claims that the disease can be quickly cured by the administration of large doses of thymol.

Professor George R. Murray and Dr. P. Thornton, in their contribution to Quinn's " Dictionary of Medicine," discussing the cause of goitre, found that goitre does not exist in Greenland or Lapland, whilst it is prevalent in Sumatra, where snow is unknown. They agree that whatever the active agent may be, its activity is destroyed by heat, as the water is rendered harmless by boiling. Although no living micro-organism has been isolated, the probability is that such is the existing cause of the disease, and possibly it may be found to be associated with some condition dependent on the existence of such an organism either in the soil itself or in the water from neighbouring high altitudes.

DISCUSSION.

Dr. R. F. Llewellyn, Braidwood, N.S.W., said: " Simple goitre " is endemic in my district. I find that in those parts of the district where there is most calcium in the drinking water goitre is most frequent. Observations extending over nearly ten years have convinced me that, while the origin seems to be microbic in certain localities, we should not yet exclude calcium finally from our minds as a causal factor. I had a remarkable experience a few years ago with a case of pulmonary tuberculosis. I gave (for reasons which it is not necessary to go into here) 5 grains of calcium chloride three times a day to the patient—a boy 15 years of age. After continuation of this treatment for about six weeks, examination disclosed the presence of a well-marked enlargement of the thyroid—although the thyroid had, previous to the initiation of the calcium treatment, been not even palpable. While it must be admitted that this may have been the result of coincidence, I think it is certainly suggestive, especially as I have never yet known an individual presenting simple goitre to develop any form of tubercular disease. Gauthier's successes with thyroid feeding for delayed union in bone seem to indicate that there is an important part played by the thyroid in the changes which calcium undergoes in the body; and on the general principle that any gland which is worked at high pressure tends to hypertrophy, I think it is fair to assume for the present that calcium is a potent factor in the causation of a proportion of the cases of thyroid enlargement.

NOTES ON A VIRULENT DIPHTHERIA CARRIER.

By Frank Allwork, M.O.H., New Norfolk, Tasmania.

D.T. (m.), aged 7 years, was first seen by me on 4th May, 1910. He was suffering from laryngeal and faucial diphtheria, and was immediately sent to the Hospital for Infectious Diseases, in Hobart, where tracheotomy was performed by Dr. Sprott. He returned to his home at the expiration of four weeks apparently cured. He had contracted the disease from a neighbouring house which he had visited unknown to his parents, and where two cases, one of which was nasal, had recently been under treatment. I saw no other cases of diphtheria in this district until 2nd August, 1910. I then saw R.McD. (m.), aged 9, who had a mild faucial attack. A swab was taken, inoculation and incubation made, and a positive result obtained. At the time I failed to trace the origin of this case. On 18th August, 1910, I was called to see R.H. (m.), aged 2 years, who was suffering from laryngitis. There was, however, no membrane to be seen, and the fauces were not unduly inflamed. Suspecting diphtheria, I administered antitoxin subcutaneously, and took a swab with which blood serum was inoculated, and after eighteen hours' incubation in a thermos flask a positive result was obtained. The case was then sent to the Infectious Diseases Hospital, where it was found necessary to perform tracheotomy. As this child was related to D.T., I suspected contact with him. This I found to be the case, and also that R. McD. had played with him though not attending the same school. I then took a swab from D.T., but got no result. I would not, however, allow him to attend school, and forwarded a swab from the throat to Dr. L. S. Miller, Government Bacteriologist, Hobart, and on 24th August, 1910, there was very poor growth, owing to the condition of swab when received, and the result was negative. On 28th August, Dr. Miller again reported very poor growth, with negative result, and suggested taking swabs from the nasal passages. This was done, although they were apparently in a healthy condition, and on 10th September, the report was: "Examination of specimen for K.L. bac. gave possible results." Remarks: "After twenty-four hours' cultivation, there was one large colony, and several very minute ones, which did not give a definite result—just a few isolated bacilli—so I cultivated for another twenty-four hours, and the colonies had much increased in size, and gave a very definite result. As to the virulence of the bacilli, I can form no idea, as we have no guinea-pigs.—L. S. Miller, Bacteriologist."

On 12th October, Dr. Miller reported on a nasal swab:—"There were some definite clumps of K.L. present, though in very few numbers." 10th November, 1910, report:—"After twenty-four hours fairly good growth of small colonies, which produced small fat bacilli (an involution form of K.L.) after forty-eight hours very extensive growth. Bacilli were larger, and no doubt of K.L." 20th January, 1911, Dr. Miller reported:—"Result negative; no definite K.L. present. I think one may be quite safe in the boy, who has ceased to be infective."

Listerine was used as a douche during the whole period the boy was under observation.

Only one other case of the disease has occurred within this district since those quoted above occurred, and it could in no way be associated with them.

The practical question arising out of the above case is: "What provision should be made for the further isolation and observation of cases of diphtheria after their discharge from the institution where they have been treated?"

Dr. J. H. L. CUMPSTON pointed out that in the recently-introduced Health Act of Western Australia, the definition of infectious disease had been extended to include carrier cases, so that these infective persons may be subjected to exactly the same restrictions as well-developed clinical cases.

A COMPARATIVE STUDY OF SOME STRAINS OF DIPHTHERIA AND DIPHTHEROID BACILLI.

By J. BURTON CLELAND, M.D., C.M. (Syd.).

Principal Assistant Microbiologist, Government Bureau of Microbiology, Sydney.

IN examining the various strains of diphtheria bacilli and diphtheroid organisms comprised in the stock cultures of this Bureau, or isolated from time to time from man or animals during our investigations, the value of the sugar reactions, as a further aid to the morphological differentiation of types, has proved of considerable value. As is well-known, the true diphtheria bacillus produces acid on glucose but not on saccharose, whilst another large group of diphtheroid organisms produces acidity in both of these media. It is the object of the present paper to present some of these results, accompanied with notes as to the origins of those strains isolated by us. The general prevalence of this type of organism in man and animals is also thus illustrated.

The Diphtheria and Diphtheroid Group of Organisms.

This group of organisms may be described as slender gram positive bacilli, often with somewhat pointed ends, tending to lie in parallel lines in culture media, and showing more or less markedly irregularities in staining by appropriate methods, as, for example, darker granules or bands. Often parallel bacilli, lying side by side, are exact copies of each other as regards the size and situation of the granules, suggesting, strange to say, the occurrence of longitudinal fission. The colonies on agar or solidified blood-serum grow slowly, and are at first small and semi-transparent and almost streptococcal-like. Later the growth becomes more luxuriant and heaped up, and may assume a certain amount of yellowish colouration.

True Klebs–Lœffler or Diphtheria Bacilli.

(TYPE A..........)

The ordinary morphological appearances of these bacilli need no recapitulation. Sown on the five common sugars, we find invariably acid on glucose, and no change on mannite, dulcite, lactose, or cane sugar. This may be expressed by the formula A.......... In addition to these results, we get almost invariably acid on galactose and no reaction on inulin, amygdalin, salicin, raffinose, sorbite, erythrite, and adonite. There is usually no change also on maltose, dextrin, and arabinose. In describing the following strains, only any departure from these reactions will be noted :—

1. (K_1)—B. diphtheriæ, virulent. Inst. Pasteur, France. Tested on the five common sugars only.
2. (K_2)—B. diphtheriæ. Old stock culture. Not tested satisfactorily on dextrin and arabinose.
3. (K_4)—B. diphtheriæ from an epidemic at Parramatta. Old stock culture. Doubtful reaction on arabinose.
4. (K_5)—B. diphtheriæ. Old stock culture.
5. (K_8)—B. diphtheriæ. American type (Kral). Morphologically, very large diphtheria bacilli with a few granules and irregular banding, and with the ends drawn out. Acid slight on galactose.
6. (K_9)—B. diphtheriæ. Old stock culture. Morphologically, rather a small diphtheria bacillus with granules and bands. Only slight acidity on glucose and galactose: doubtful results on arabinose.
7. (K_{10})—B. diphtheriæ. Old stock culture. Morphologically a moderate sized diphtheria bacillus with granules. Reactions like 6 (K_9).
8. (K_{14}— Lab. 3,558)—From a case of diphtheria. Typical medium-sized granular diphtheria bacilli. A doubtful reaction on maltose.
9. (K_{18}—Lab. 3,583)—From a case of a filmy membrane on the conjunctiva, but no other evidence of diphtheria. Morphologically like a small delicate diphtheria bacillus; longer than true Hoffmann's bacillus, with occasional small granules at the ends or throughout. Not tested on dextrin, salicin, raffinose, sorbite, or inosite.
10. (Lab. 5,268)—From a case of diphtheria at Young. Morphologically, a typical granular diphtheria bacillus. After several days, an abundant raised white growth on agar.
11. (Lab. 5,770)—From the throat of a fatal case of untreated diphtheria, taken *post-mortem*. Morphologically, a typical diphtheria bacillus. A guinea-pig, inoculated with pure culture (1 cc. of a 48-hour broth culture) died in three days, whilst a control animal protected with antitoxin survived.
12. (Lab. 5,771–2)—From a case of diphtheria in the Royal Alexandria Hospital for Children. This culture was submitted for comparison with cultures taken from wounds in the Hospital, which showed the presence of diphtheroid bacilli. These are described under the latter heading. Morphologically a typical diphtheria bacillus. Inoculation into a guinea-pig caused death in two days, whilst a control animal protected with antitoxin survived.
13. (Lab. 6,059)—A culture submitted for examination by us as to lethality. Morphologically, it appeared, in a 24-hour serum culture, as short, thin, irregularly stained bacilli, usually with a break in the centre; there were also longer forms up to

enormous involution forms with dumb-bell-like knobbed ends. Granules were occasionally present but were not numerous. In later subcultures, the organism appeared as a typical banded diphtheria bacillus without prominent granules. Tested on sugars, the organism showed a departure from type in giving acid in dextrin. Two guinea-pigs were inoculated with a pure culture, one being protected with antitoxin—both died in 5 days; a third guinea-pig, inoculated as before and not protected with antitoxin, survived. The deaths of the first two guinea-pigs were probably accidental, and not referable to the injections. The organism possessed evidently little, if any, toxicity.

14. (Lab. 6,483)—From a convalescent from diphtheria, Sydney. Morphologically, this was a small diphtheria bacillus with irregular staining and granules not prominent. This type persistently gave acid in maltose.
15. (Lab. 6,591)—From a case of diphtheria in a child of 6 years, Sydney. Morphologically, a typical large granular diphtheria bacillus. Gave acid on dextrin.
16. (Lab. 6,671)—Culture from the throat of a medical man in a hospital. The organisms had been present for over a week, but produced no inconvenience beyond a little soreness and redness of the fauces. Morphologically, a typical large granular diphtheria bacillus. This culture gave some acidity on saccharose in five days. Of two guinea-pigs inoculated with cultures, the one not protected with antitoxin died in six days.
17. (Lab. 6,936–1)—From a case in the Royal Alexandria Hospital for Children. Morphologically, a typical diphtheria bacillus with small granules.

The next organism, though morphologically similar to and giving the same reactions as the true diphtheria bacillus, was probably not that organism. It was obtained in abundance from skin lesions on a sheep, and was doubtfully lethal to a guinea-pig.

18. (Lab. 7,442)—This organism was present in the crusts from a case of necrosing dermatitis of the face and ears of a sheep near Bathurst (January, 1911). It was evidently an adventitious organism, since it has not been obtained from other cases of this disease since examined. The organism grew as minute white colonies on agar, more luxuriantly on blood-serum. The bacilli were Gram positive, showed granules by Neisser's stain, and could barely, if at all, be distinguished from true diphtheria bacilli. By an independent examiner, who did not know their origin, they were considered to be typical diphtheria bacilli. The following inoculations were performed:—A guinea-pig was inoculated with a 48-hour broth culture, and died at the end of nine days with no lesions visible at the site of inoculation, but with congested lungs—the organism was not recovered from the site of inoculation. A second guinea-pig, similarly treated, but given 1 c.c. of antitoxin as well, remained well. The culture was also inoculated by scarification on the side of the face and nostrils of a sheep. This was followed by slow scabbing, which disappeared in a few days, without the production of lesions similar to the original condition.

Diphtheroid Organisms.

(TYPE A..........)

These diphtheroids, which morphologically, or for other reasons, were not true diphtheria bacilli, gave the reactions of true diphtheria bacilli on the first five sugars.

19. (Lab. 7,828)—This organism was grown on blood agar from a case of urethral discharge, together with Gram negative micrococci. It appeared as minute colonies of Gram positive diphtheroid bacilli. Of four subcultures made on the first five sugars, in three acid appeared in glucose (in one of these slowly), and in one there was no change. The organism died out before further tests were conducted.
20. (Lab. 7,646)—Numerous diphtheroid bacilli were isolated from the milk of a cow (Brooklet, February, 1911), apparently suffering from contagious mammitis. The bacilli were small and Gram positive. They showed barred forms like diphtheria bacilli, but granules were few. On agar there was a fine white growth, and on blood-serum it was more abundant and whitish to (later) pale orange. There was only a doubtful slight acidity on glucose in ten days.

(TYPE A.........A.)

The organisms of this type are usually smaller than the true diphtheria bacillus, rarely show granules, and usually possess a lighter-staining central area. The reactions on glucose and saccharose are acid, with no effect on mannite, dulcite, and lactose. Acidity is usually produced on galactose, but not on the other sugars previously mentioned. Departures from this type will alone be noted.

21. (K_{16})—Diphtheroid bacillus from a plague bubo. Morphologically, a typical small granular diphtheria bacillus.
22. (K_{17})—Diphtheroid bacillus from throat. Morphologically, a small diphtheroid like Hoffmann's bacillus with occasional granules at the ends or rarely in the middle.
23. (K_{20})—Diphtheroid bacillus from ulcer of skin. Morphologically, a small diphtheroid with occasional granules like diphtheria bacilli. Doubtful reaction on arabinose.
24. (K_{22})—Diphtheroid bacillus from an abscess. Morphologically, a medium-sized diphtheroid bacillus without granules.
25. (K_{23})—Diphtheroid bacillus from an ulcer in the neck (Hay). Morphologically, like a small diphtheria bacillus, with fairly frequent granules not present in all the bacilli.
26. (K_{24}—Lab. 3,303)—A diphtheroid bacillus associated with actinomycosis of the neck in man, both being cultivated from the same pus. The patient has greatly improved, perhaps as the result of a vaccine of the actinomycosis which was used. Morphologically, the organism was a typical small granular diphtheria bacillus.
27. (Lab. 5,771, a and b)—On several occasions, at the Royal Alexandra Hospital for Children, diphtheroid organisms have been isolated from surgical wounds in patients not admitted for diphtheria and not in the same wards as these cases. One, if

not more, sudden deaths in such cases were attributed to such infection. These cultures, for which we are indebted to Dr. Stewart, represent the organisms from two of such wounds which were submitted, together with the throat culture (*see* No. 11, Lab. 5,771–2) from a true case of diphtheria. Morphologically, by the sugar reactions, and by the lethality, the two types of culture were readily identified. Morphologically these diphtheroids were smaller than true diphtheria bacilli, and showed irregular staining with a lighter centre and pointed ends, and only occasional granules with Neisser's stain. The growth on agar at the end of seven days also showed decided differences. Thus the true diphtheria bacillus strain showed a growth more filmy, less densely white and less raised than the other, which was a dense, white, raised moist growth. One of these diphtheroid cultures conformed exactly with the above-given sugar reactions, the other in addition yielding acid on maltose. Guinea-pigs inoculated with each of these cultures (four animals in all, two receiving antitoxin as well) all survived, whereas, as already mentioned, the guinea-pig inoculated with the true diphtheria bacillus and not protected, died in two days. From a third surgical case, a similar diphtheroid, not tested on animals, was also obtained.

28. (Lab. 6,936–2)—This diphtheroid organism was received from a surgical case in a boy at the same hospital as No. 27 (Lab. 5,771) came from, but at a later date (December, 1910). The organism appeared to be identical with the preceding ones from this source. On agar, it grew abundantly as a dense white growth. Two guinea-pigs were inoculated in the usual way, each with 1 c.c. of the forty-eight-hour broth culture, one being protected wth antitoxin: the protected animal died in seven days, and the unprotected one in twelve days. It would seem to be doubtful, therefore, whether the inoculations were responsible for these deaths.

29. (Lab. 6,302 and 6,867)—These two strains, which proved to be identical, were isolated on different occasions at intervals of several weeks from a lumbar sinus in a girl of 20, which followed on an abscess about a year before. The colonies on agar were moderately large and whitish, later showing knobs on top. Stained with Neisser, the organism appeared as a small diphtheroid, in shape like a diphtheria bacillus. Granules were very few and small, when present, being at the ends or throughout; the protoplasm stained irregularly, showing a break in the centre; in some cultures, there were no granules. Two guinea-pigs were inoculated in the usual way; the one protected with antitoxin died in ten days, the other in thirteen.

The Diphtheroid Organism in General Paralysis of the Insane.

(Type A—A—A.)

Through the courtesy of Dr. Flashman, I have also had an opportunity of examining a culture of a diphtheroid isolated from the bladder of a case of general paralysis of the insane by Drs. Flashman and Latham. This on agar gave a moderate growth of small colonies thickening to a very pale fawn white, and on solidified blood-serum a more abundant growth of pale fawn white. The organism has only been tested on five sugars, yielding acid on glucose, *dulcite*, and cane sugar, but none on mannite and lactose. This reaction on dulcite, if constant, is an important item in identification.

SUMMARY OF RESULTS.

Diphtheroid organisms have been obtained from a variety of lesions in man and animals. These include surgical wounds and sinuses (several occasions), a plague bubo, ulcers, an abscess, in association with actinomycosis of the neck, and from an urethral discharge in man; in animals, from necrosing dermatitis of the head of a sheep and from the milk of a cow. Most of these diphtheroid bacilli could be readily distinguished morphologically and by their sugar reactions from true diphtheria bacilli. In one instance, that from a sheep, there was practically no difference between the organism and a non-toxic diphtheria bacillus.

I am indebted to the courtesy of the Director of the Bureau of Microbiology (Dr. F. Tidswell) for permission to publish these results.

A PRELIMINARY REPORT ON THE BACTERIOLOGY OF FÆCES IN GASTRO-ENTERITIS.

By E. JEAN DALYELL, M.B., C.M. (Syd.).

Junior Demonstrator in Pathology, University of Sydney, from the Pathological Laboratory, University of Sydney.

THIS investigation was carried out during the winter months of 1911, from May to August, on sporadic cases of enteritis and gastro-enteritis which had been admitted to hospital. Thirty-six specimens of fæces were examined, collected from twenty-four patients. Specimens from the same patient were examined, in some cases, on two or more occasions.

The procedure followed was that described by Morgan in his account of the investigation of the organisms from cases of summer diarrhœa in infants (*British Medical Journal*, 1906–1907).

i. An emulsion was made of a portion of the fæces in bile-salt broth.

ii. After two to six hours bile-salt lactose-agar neutral-red plates (MacConkey) were inoculated from this.

iii. After twenty-four hours, colourless colonies were picked off the plates, and cultured in Durham's tubes containing lactose.
After incubation for four days, all tubes showing gas formation were rejected.

iv. Non-lactose fermenters were further investigated.

The twenty-four cases examined were all between the ages of one month and fifteen months.

Three were surgical cases with normal stools, and were investigated as control cases.

I failed in two cases of the remaining nineteen, to isolate any non-lactose fermenters; so that the organisms investigated represented twenty-two cases.

Forty non-lactose fermenters were classified, by means of their cultural reactions in litmus milk and in media containing glucose, mannite, dulcite, lactose, saccharose, maltose, dextrin, inulin, raffinose, sorbite, galactose.

By this means I found in seven different cases a short mobile bacillus which produced permanent alkalinity in litmus milk; formed acid and gas in dextrose and galactose, and did not affect any other sugar media.

This therefore conforms to the type known as Morgan's No. 1 bacillus, described by him in 1906.

Agglutination tests were tried with the serum of three of these patients (diluted 1 : 60), against their own and two other Morgan's No. 1 bacillus. The results were negative in every case.

In four cases an organism was isolated which formed acid and gas in glucose, mannite, dulcite, maltose, sorbite, galactose, and did not affect the other sugar media.

Three of these produced permanent acidity in litmus milk, which characterises the paratyphosus A. The other caused alkalinity in litmus milk, so I have classed it as paratyphosus B, or else B. enteriditis.

In the appended table the cultural reactions are fully shown.

It is not possible to base conclusions on so small a series, in the absence of an extended series of controls; but the fact of finding Morgan's No. 1 bacillus in seven cases out of twenty-one cases of gastro-enteritis is of interest, as I have found no previous account of similar findings in this State.

The investigation is proceeding with a view of determining the incidence of this organism during the summer months; and the effect of vaccine therapy on the condition.

Dr. Margaret Harper, of the Royal Alexandra Hospital for Children, and Dr. Schlink, of Royal Prince Alfred Hospital, have kindly allowed me to collect material from the wards; and I am indebted to Dr. Bradley, for stock cultures of Morgan's No. 1 Bacillus, from the Bureau of Microbiology, Sydney.

CASE NOTES.

A. $\frac{9}{12}$. Chronic catarrhal colitis. Recovery. Morgan's No. 1 found on two occasions (three weeks interval between examinations).

B. $\frac{6}{12}$. Acute inflammation of bowel and stomach. Death.

C. $\frac{2}{12}$. Ill from birth. Had great variety of organisms. Morgan's bacillus No. 1 found, *B. paratyphosus*, A., found on two occasions (fourteen days between examinations). Death.

D. $\frac{3}{12}$. Acute inflammation of stomach and bowel. Death.

E. $\frac{7}{12}$. Acute colitis. Recovery.

F. $\frac{15}{12}$. Chronic gastro-enteritis. Death.

G. $\frac{5}{12}$. Acute inflammation of bowel. Death.

H. $\frac{7}{12}$. Chronic catarrhal colitis. Recovery.

I. $\frac{5}{12}$. Chronic inflammation bowel and stomach. Death.

J. $\frac{2}{12}$. Ill from birth. Catarrhal inflammation of bowel. Death.

Case.	Morphology.	Motility.	Litmus milk.			Glucose.	Mannite.	Dulcite.	Lactose.	Cane sugar.	Maltose.	Dextrin.	Inulin.	Raffinose.	Sorbite.	Galactose.
			1 day.	3 days.	15 days.											
A, B, C, D, E, F, G	Bacillus	M	O	Alk	Alk	AG	...	...	...	...	...	...	...	...	...	AG
C, H I	Bacillus	M	ac	ac	ac	AG	AG	AG	...	...	AG	A	...	...	AG	AG
J	Bacillus	M	ac	alk	alk	AG	AG	AG	...	...	AG	A	...	...	AG	AG

HYPERNEPHROMATA—THEIR PATHOLOGICAL ASPECT.

By Sydney Jamieson, M.B., C.M. (Edin.).

In a paper which I have prepared for this section of medicine, I have drawn attention to the peculiarities of these growths in their clinical aspects.

I now wish to describe their pathological anatomy and histology, and having done so will say a few words on their pathogenesis.

(I) Morbid Anatomy.

So characteristic are the naked-eye appearances of these growths that anyone who has studied them can almost, with absolute certainty, not only diagnose them as soon as they are exposed by section, but predict the appearances that will be seen on microscopic examination of their various parts.

One of the most striking features of these growths is their tendency to assume a more or less definite encapsulation; and yet, pathologically speaking, they have no true capsule.

From the kidney substance the line of demarcation between growth and organ may be fairly distinct, and they may be even partially separable from the substance of the kidney itself.

So much is this the case that a surgeon may be tempted to proceed to enucleation of the growth; experience, however teaches that such treatment is most unscientific.

Another feature of these growths is, that they grow out from the kidney-substance and leave a large part of the organ apparently normal.

Sooner or later in most cases the tumour breaks through its false capsule, and extends in a diffuse manner through the pleinephoric fat.

Commonly the tumour appears as a smooth nodule or bossy mass projecting from the surface of the kidney, and when the growth is large, as it often is, the kidney may appear as a mere appendage to the growth.

In its outward appearance, as a rule, the growth is of a yellowish-white colour.

In consistence it varies, in some parts being firm and somewhat elastic, in other parts it may be semi-fluctuant. In some cases large cystic areas are to be found.

It is, however, on section that the most characteristic features are to be noted.

The first thing that strikes one is the striking absence of homogeneity that appears.

The growth is cut up into lobules of varying sizes by slightly marked fibrous bands.

The greater part of the section is for the most part of a yellowish-white colour, opaque, and apparently structureless.

Some parts, however are more translucent and of greyish-white appearance, while others show softening and an appearance suggestive of caseation.

The most characteristic feature, however, is the occurrence of patches of varying size of a bright, often ochre-yellow colour, resembling somewhat the appearances seen in deep-seated gummata in the liver.

They differ, however, from gummata in being irregular in shape and in not being so tough in consistence.

In addition to the characters already mentioned, there are often areas of reddish or brownish colour with a more fleshy aspect.

In some cases the process of necrobiosis which has taken place, has resulted in the formation of large cystic spaces, and in the walls of these cysts it is not uncommon to find gritty masses of calcareous material.

Very frequently the pelvis of the kidney is pressed upon and displaced by the growth, and occasionally, though this is rare, the growth eats its way through into the pelvis.

Another feature of these growths (and this one is by no means uncommon), is that they invade the renal vein, and this sometimes at an early stage.

The most important points, then, in the anatomy of these growths are—

1. Their apparent encapsulation.
2. Their sharp delimitation from the kidney-substance.
3. The striking want of uniformity of the appearance on section.
4. The occurrence of caseation and sometimes of cysts.
5. The occurrence of bright yellow areas.
6. The early invasion of the renal vein.

These, then, are the chief anatomical features, and so distinctive are they that by them alone an accurate diagnosis of the nature of the growth may be arrived at.

(II) Their Histology.

The most important point in connection with the microscopic anatomy of these growths is the curious variation in the structure of the tumour in different parts.

This variation is seen, not only in the character of the cell elements of the growth, but also in the arrangement of the tissue.

Such diversity is seen that these growths have been variously classed as adenomata, adeno-carcinomata, sarcomata, endotheliomata, and a host of other names have been applied to them.

These mistakes have arisen from the habit of reporting on their nature from a single section from one separate part of the growth, and can only be avoided by taking sections from several parts

The most important point, then, in regard to the histology of these growths is the variation in their structure in different parts of the growth.

The Cell Elements of the Growths.

The growth is for the most part made up of cellular units, and the stroma, such as it is, is both scanty and irregularly disposed.

The cells show remarkable disparity as regards size and form, and they differ much as regards their action to stains.

Roughly speaking, in the great majority of examples of this growth, the cells may be described as belonging to three types—

1. Clear swollen cells.
2. Dark granular cells.
3. Giant cells.

1. *Clear swollen cells.*—This is by far the most common type of all seen. They are large and globular. Have a distinct outline with a distinct and well-marked nucleus. The cell body is clear and stains but sparingly.

These cells are usually tightly packed together, and with their sharp outlines produce a picture which resembles somewhat a mosaic.

2. *The Dark Granular Cells, or as they are sometimes called "opaque" cells.*—These show up by sharp contrast with the clear cell. Their protoplasm is granular and stains deeply. The nucleus, though distinct as a rule, is sometimes obscured by the dark staining of the cell.

These cells are usually smaller than the clear cells, have angular outlines, and are not tightly packed together but often loosely arranged.

Both these types of cell (1 and 2) often occur together in one and the same microscopic field, but as a rule one or other is more numerous in any given field.

3. *Giant cells.*—Although on the whole the clear cells are larger than the dark cells, yet it is among examples of the dark variety that giant cell formation is seen.

These giant cells, in some tumours, form quite a distinctive feature and are quite numerous, but this is not uniformly so.

Some of these cells attain to a great size, and occasionally plasmodia-like masses are seen.

These giant cells, as a rule, take stains readily, but in most instances the central part stains more deeply and is surrounded by a zone of cell protoplasm which stains more faintly.

Some of these giant cells are multinucleated, but by no means infrequently it is found that what at first sight appears to be nuclei are in reality englobed polymorpho-nuclear cells. Some of these englobed cells show signs of disintegration, while others stain sharply and definitely and appear to be surviving within the plasmodial masses.

In addition to these three types of cells, some sections show large round masses of regular outline with pale outlines and darker centres.

These masses often show no nuclei.

Most histologists regard these giant cells as being due to the enlargement of individual cells and not to the fusion of adjacent cells.

This view is borne out by the fact that not uncommonly groups of cells are found arranged around a blood vessel, and one or more of these have enlarged so as to form club-like masses projecting outwards from the main mass.

So much, then, for the cell elements. Now let us study—

The arrangement of the cell elements.

The main feature in this connection is that the cells are arranged in close relationship to thin-walled blood vessels.

This perivascular arrangement of the cells is very characteristic.

Between the cells themselves and the blood vessel, however, there is usually a scanty amount of connective tissue.

A study of the arrangements seen reveals two main types of arrangement, viz. :—

1. A solid alveolar type.
2. A tubular perivascular type.

1. *The solid alveolar type.*—In some parts this arrangement resembles that seen in highly cellular carcinoma.

For the most part this type has a vascular stroma, consisting of a narrow vessel with thin walls, and surrounded by a small amount of connective tissue.

It is in these parts that the resemblance to an adeno-carcinoma of the thyroid is seen, viz., a cellular mass with a stroma made up of blood-vessels.

The alveoli are mostly small, their contents being solid and closely packed.

In some parts the alveoli are larger and the cells less tightly packed. In this case the cells are found close to the vessels and the rest of the space more or less empty—an arrangement which seems to admit of the cells growing and forming the club-like masses described.

Degenerative changes are frequently seen among the cells of the alveoli, and this is most prone to occur towards their centres, and when the process is advanced the alveolus shows a central area of protoplasmic and nuclear debris surrounded by a peripheral zone of healthy cells several layers thick.

This appearance closely mimics that seen in tubular carcinoma of the breast.

2. *The tubular perivascular type.*—Here the structure resembles that seen in a columnar-celled carcinoma.

In this type long ramifying spaces are seen, lined to a large extent by a single layer of well-formed cubical or columnar cells.

From the walls of these spaces papillary prolongations are often seen, such as are commonly to be noted in the cystic adenoma of the ovary.

The stroma of this type consists of thin-walled blood vessels coursing among the tubules.

The cells in this type are smaller and more regular than those seen in the alveolar type.

(III) Their Pathogenesis.

Ever since these tumours were first accurately described by Grawitz some twenty-six years ago, the views enunciated by him as to their origin have been generally accepted as correct.

The views he propounded were, that these growths depended upon the abnormal growth of remnants of suprarenal tissue so commonly found in the kidney.

The hypothesis of Grawitz was, however, challenged in a masterly fashion in 1908 by Stoerk, who maintained that these growths are really renal in origin.

In favour of his views, Grawitz brought forward the following statements :—

1. The situation of these growths in the sub-capsular region of the kidney corresponds with that of the suprarenal islets found in the organ.
2. The marked distinction between the character of cells of these growths and those of the renal parenchyma.
3. The fact that fat is found in the cells of these tumours and also in the cortical cells of the suprarenal body, while it is not a normal constituent of the renal epithelium.

Stoerk, on the other hand, asks the question, " Why do hypernephromata always originate in the kidney and never in the suprarenal? "

This statement, if true, would suggest a powerful argument against the views of Grawitz, but that it is not, at any rate universally, true, I can prove by the fact that about a year ago I met with a tumour growing from the left suprarenal which showed all the characters, both macro and microscopic, of an ordinary hypernephroma, and yet it was in no way connected with the kidney.

The growth in this case showed an extensive metastasis of the pleura of the base of the left lung.

Another statement made by Stoerk in this connection was to the effect that growths of the suprarenal, both benign and malignant, are always very different in structure from the hypernephromata.

The growth I describe, on the contrary, does show all the characters of the hypernephromata.

Another argument, and one which I think must carry considerable weight in favour of Stoerk's views, is that although islets of suprarenal tissue are often found in the liver, yet hypernephromata have not been described in that situation.

He further adduces the fact that, while suprarenal islets are most common at the upper pole of the kidney, hypernephromata are most commonly situated in the central region of the organ.

Moreover, he indicates that although suprarenal islets may be found within the substance of the fibrous capsule of the kidney, this is never the case even in the smallest of hypernephromata.

He also points out that the "swollen" cells which form so characteristic a feature of these growths are quite unlike any cells found in the suprarenal body.

An argument suggested by Trotter, and one which must carry considerable weight, is one founded on the analogy, as regards complexity of structure, that exists between the tubular structure of the kidney and that of the testicle.

Both these organs maintain their function till late in life, and the growths affecting both these organs are remarkable, both for their complexity and multiformity.

Such complexity of structure is not to be found in the case of the suprarenal body, and this constitutes a strong argument against the views of Grawitz.

Such, then, is the present position of the views as to the pathogenesis of these growths, and it must be admitted that the discussion of the subject still leaves much to be proved before a complete consensus of opinion on this matter can be arrived at.

In conclusion, I should like to point out the resemblance that exists between the structural peculiarities of the hypernephromata and that remarkable growth of the uterus—the chorio-epithelioma.

These two growths resemble one another in three characteristics:

1. In the cells of both it is not uncommon to find droplets of a highly refractile character resembling fat.
2. Both exhibit areas in which large masses of multinucleated protoplasm are found.
3. In both the cell elements of the growth show a very clear relationship to the blood vessels; and in both the growths by no means infrequently invades the lumina of the blood vessels.

ENDOTHELIOMATA AND ALLIED TUMOURS.

By Alfred E. Finckh, M.B. (Syd.).

It will go without saying that, for anyone in practice, even if he be a pathologist, much original work on so intricate a question as the tumours, known as endotheliomata, must be out of the question.

The object of this paper, therefore, is to state what the generally adopted ideas of the terms, variously applied to such tumours, are, and to create discussion as to what extent it is advisable to use such terms, especially from a practical point of view—that is, from the point of view of the pathologist who is called upon to give a name to a tumour, in order, thereby, to indicate to the surgeon what treatment is to be adopted.

The whole question of the nomenclature of these tumours is still one that gives to the clinical pathologist a good deal of anxiety and worry when a definite diagnosis is required, a diagnosis which is to allow of an intelligent interpretation by the surgeon. The busy surgeon is not in a position to criticise pathological nomenclature; if a certain tumour is diagnosed by one pathologist as a lymphangiosarcoma, and by another as an endothelioma, he finds himself in a somewhat difficult position, for it may require on his part a good deal of looking up of literature before he discovers that both pathologists meant the same thing.

It is from the point of view of giving to the surgeon the exact knowledge, which the surgeon is desirous to have, when he sends a tumour to the pathologist, that the writer would like to see more co-ordination between the histological findings of the pathologist and the clinical finding of the surgeon. The pathologist is apt to be too academic when giving a diagnosis, and the more difficult the case the more academic is he liable to become, and the more doubtful does it, as a rule, leave the surgeon. The information the surgeon requires is not so much as to the exact nature of the growth, but rather in regard to the steps he should take in order to eradicate the disease. To him it is of much greater value to be told that, in a particular case, he should remove the growth with as wide a range as possible, than to have an elaborate description of the microscopical finding of the tumour.

If we consider that the endotheliomata offer histologically greater difficulties in diagnosis, and have therefore given rise, more than any other class of tumour, to conflicting controversies, it is easy to understand that there should be this lack of uniformity as regards nomenclature. Nor does it appear from the nature of the controversial points at issue that, in the near future at least, we are likely to see much clearing up in the matter.

On the other hand, if the pathologist is to work hand in hand with the surgeon, what should be aimed at is a nomenclature which, to the surgeon, has some definite meaning, and which carries with it to the mind of the surgeon a definite meaning as regards recurrence, metastasis, and malignancy.

Endotheliomata are tumours which have arisen from the tissues known as endothelium. These cellular elements are usually spoken of in text-books on histology as pavement epithelium, occurring on internal surfaces, such as those of the serous membranes, blood-vessels, and lymphatics. However, the meaning of this term is by no means so simple. Some histologists give the term a very wide range, and include in it the cells lining the pleura, the peritoneum, and the free surface of dura and pia mater; others, however, will have the term confined to such cell elements which, from a genetic point of view, are to be classified with connective tissues. This definition would make it doubtful as to whether any but the lining membranes of lymphatics and blood-vessels, and the covering of the brain-membranes, are to be defined by the term, and brings us at once into the great conflict of having to decide for this classification between epitheliomata and sarcomata. The cells lining serous cavities are, it is true, continuous with those lining the lymphatics, but such continuity is only secondary, and in their embryonic period they have been shown to possess definite characteristic features which will not allow of their being classified with the abovenamed endothelial cells. The fact that, in inflammatory processes of the peritoneum, these cells occupy a position of their own, and do not take a share in the formation of connective tissue new growths, goes to prove that during their earliest differentiation they are closely allied to epithelial cell elements.

Hence we find that the term endothelium in itself is by no means easy of definition; how much greater, then, must be the difficulty of classifying tumours supposed to have arisen from them.

When we examine a tumour under the low power, our attention is drawn to the possibility of a diagnosis of endothelioma by the presence of masses of epithelioid cells, rich in protoplasm, which cover surfaces, line cavities, or completely fill them. Intervening between these we get connective tissue stroma with vascular elements. Under the high power it will be seen that intercellular substance may intervene between the cell elements, thus resembling connective tissue growths, or there may be a total absence of intercellular tissue, so that in one instance the growth may be sarcoma-like, whereas, on the other hand, it resembles a carcinomatous growth. The cell elements vary greatly in size and shape, so also the nuclei. A peculiar degeneration of the tissue, which may be mucoid, hyaline, or amyloid, is said to be characteristic of these tumours, but in only a few of our specimens does it become apparent. On the whole, tumours may show an enormous variety of features as to be on the one hand remarkably like the alveolar type of carcinoma, whereas, on the other hand, the intercellular connective tissue may render it almost indistinguishable from a true sarcoma.

Together with the endotheliomata, it is usual to classify such tumours as Peritheliomata, Cylindromata, Psammomata, and Cholesteatomata.

As regards peritheliomata, the difficulties which arise are still greater, for here we are attempting to apply to a class of tumours a specific name derived from elements which are still mystical, for neither developmentally nor physiologically has the so-called perithelium a right of existence, and not even normal histology is prepared to grant it a definite place in the classification of tissues.

Since the tumours thus spoken of as peritheliomata seem to arise in the perivascular lymph spaces, it seems best, until the term perithelium has become better established, to classify them as endotheliomata of the perivascular spaces.

The so-called cylindromata are another group of tumours which have not yet been sufficiently clearly defined to justify their classification amongst this, or any other group of tumours. The name of these is derived from the fact that, as a secondary component, the tumours are made up of hyaline masses which may be cylindrical, or may take the shape of branching strands or of isolated, club-shaped or spherical masses. These are surrounded by layers of cells usually more than one thick, thus having a strong resemblance, when examined under the low power, to the endotheliomata. On the other hand, there may be present masses of cells without enclosed hyaline structure, and in this form such tumours closely resemble carcinomata, but differing from these, inasmuch as such cell elements, together with the connective tissue in the tumour proper, advance uniformly in growth as a closed tumour, and do not infiltrate the neighbouring tissues. Microscopically, there is nothing to indicate that these cell elements have been derived from endothelial or epithelial tissue, and, so far as the diagnosis is concerned, it purely depends as to whether we are to follow the classification of Borst, who groups them together with the endotheliomata, or with Ribbert, as a distinct class by themselves, or with Lubarsch, who names them carcinoma cylindromatosum.

The psammomata and cholesteatomata are of the same histogenetic uncertainty, and need only be mentioned here because they are members of the same group.

I must confess that I consider this paper as most disappointing. I took the matter up on account of the fact that during the last four or five years Dr. Jamieson and myself have felt justified in diagnosing, at the Pathological Laboratory of the Sydney Hospital, some seventeen tumours as endotheliomata. I was full of enthusiasm when I started, but the deeper I dug into the literature the deeper confusion seemed to become, until at last I had to give up entirely the idea of being able to throw any light on the subject which would have assisted me in giving a more definite prognosis to the surgeon.

On the other hand, I am more than ever convinced that, from a clinico-pathological point of view, there is much to be done in clearing up this somewhat difficult question, at least to the satisfaction of the surgeon. What the surgeon inquiries after is: To what extent are these tumours malignant; to what extent do they recur locally; and to what extent do they form metastatic growths? Not to any of these questions even the minutest histological investigation gives an answer.

Of our Sydney Hospital cases two were fatal. Both of these were endotheliomata of the pleura, with metastases in the other organs. All the other cases, with the exception of one, which was a small growth in the thigh, were tumours from regions of the head and neck; four of them were from the parotid.

In several of the cases operated upon there was a local recurrence, but on the whole malignancy was of a mild type, and without metastasis. This seems to agree with the generally expressed opinion as regards their malignancy. Some pathologists, indeed, doubt if endotheliomata ever give rise to metastatic growths, and refuse to accept such tumours as endotheliomata, but look upon them as a tubular type of carcinomata. This especially refers to the endotheliomata of the pleura.

Thus there is still much left to be done as regards these tumours, and, if I may be allowed to do so, I would suggest that this question be put on the business-sheet for general discussion at the next Congress. If, during the next two years, an attempt were made to collect, with the collaboration of our surgeons, all possible data as regards these tumours, I have not the slightest doubt that a good deal of valuable light would be thrown on this complex subject. I would propose that in each of the States and in New Zealand a small sub-committee be appointed, in order to bring the matter in a business-like fashion before the next Congress.

THE PARASITOLOGY OF GRANULOMA PUDENDI.

By Cecil S. Strangman.

In the discharge from lesions in this disease some twenty bodies can be seen microscopically, which differ much in form and internal structure. In old cultures, secondary fungi are often found which, on subculture on fresh media, evolve each of these bodies in their redevelopment to maturity. These secondary fungi can be grown by a symbiotic cultivation of two primary fungi which have not been isolated from the diseased parts, but are found on the skin surrounding them. Both of these organisms, during their stages of development, differ in their Gram-staining properties and methods of spore formation. At certain stages they exhibit active motility and pigment formation; the latter is always of a brown colour. When grown separately, as each form assumed reaches maturity multiplication ensues, and later on, when certain stages are reached. no further growth takes place. When an active strain of each multiplying body is obtained in pure culture the remaining stages of development are evolved, the earlier forms never being produced.

Certain large mononuclear cells, which are arranged in groups, have been described by many observers as a histological characteristic of this disease. Enclosed in their protoplasm, the organism evolves the greater part of its life-history. When free in pus many of these cells, when infected, become surrounded by a fused mass of phagocytes, which reduce them to a brownish-yellow detritus.

Clinical experience points to the probability that contact with an active open lesion, or the skin surrounding it, is sufficient to originate an infection on any of the moist parts of the pudenda. I have known a female with a single left groin lesion initiate a similar ulcer on the right groin of a male in both of whom no other parts were affected. About fourteen days after contact a papule appears, which later breaks down, forming a sore, which is covered with soft, pink granulations. As it enlarges its secretions—which leave a brown colour on dressings—ooze over the surrounding skin, on which immature fungi and spores are deposited, which originate further

SCHEME OF ROTATION
IN WHICH THE BODIES APPEAR IN CULTURES

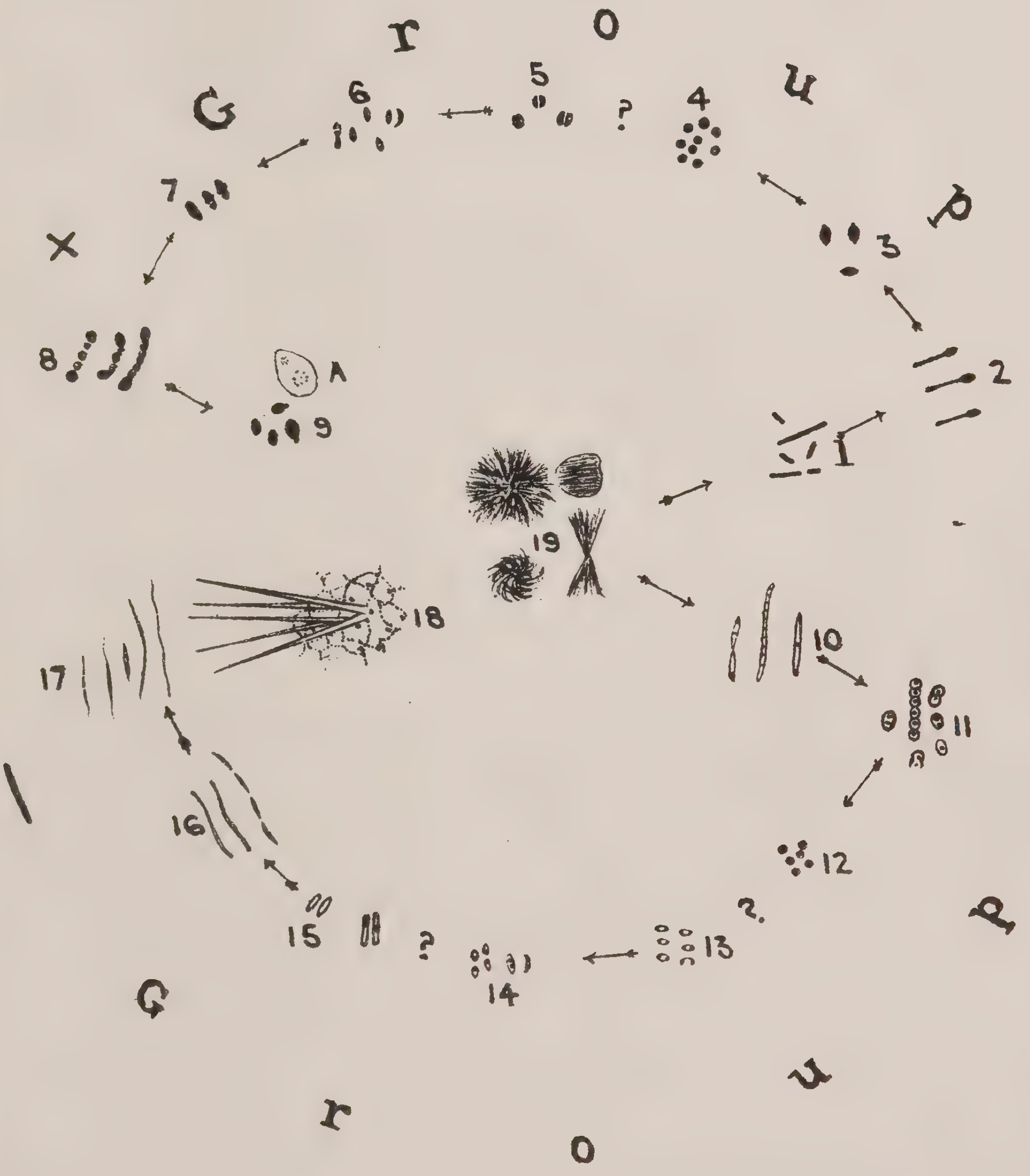


PHOTO-LITHOGRAPHED BY W. A. GULLICK, GOVERNMENT PRINTER, SYDNEY, N.S.W.

lesions on the more dependent parts, where secondary and later contact foci develop. Many secondary lesions, which contain only one group of organisms, cicatrise and heal; whilst others, after a quiescent period, show infection with the other group, when they quickly light up and spread. Fistulous tracks form in the deeper parts of the sores, which slowly dissect their way between the more resistant tissues, forming pockets which are filled with soft, fine granulation tissue. As the disease advances, stages of rest and repair alternate with periods of activity and spreading. When examining pus films from these small sores it was noticed that at times all the organisms present stained by Gram's method, and a few weeks later almost every rod, sphere, crescent, or spindle was Gram negative, and that contiguous sores varied in this respect. In small early infections the Gram positive group was generally supreme, but alternation always took place before further expansion occurred.

When the analine dyes were used for section staining, a few deep staining spores were occasionally seen lying on the stratum corneum; otherwise little assistance was gained by the study of sections from lesions with an external opening, but in deep subareolar metastases they showed the presence of clusters of spores, which are surrounded by an enveloping membrane, and groups of round cells filled with flat yellow discs, which are not of a hæmosiderin nature. Similar yellow discs are frequently met with in over-mature cultures of the secondary fungus, where they lie entangled in its mycelium. As they differ only in colour, and will not develop on plates or sub-cultures, they are probably a degenerated form of certain transparent plaques, which seem to play an important role in the life-history of the primary fungi. Sections stained by Levaditti's method often show minute, immature, intracellular spores, which retain the silver stain after prolonged treatment with Lugol's solution.

When scrapings from the skin surrounding untreated diseased parts are diluted with the water of condensation of Lœffler's medium, circular or fan-shaped tufts or sprays of transparent rods, with pointed or square ends, are seen; these rods radiate from a common centre, often giving them a mop-head appearance. When plated out, creamy-white, yellow-felted, or clear opalescent colonies are soon formed around them. A few days later these tufts and sprays, which are situated in the centre of the colonies, degenerate to a yellow, and later a brown, mass. Some of the colonies assume a dull-yellow colour, and others disappear, a faint flower-like margin only remaining. The colour and shape of these colonies vary considerably, according to the stages of development attained by the organisms at the time the plates were made. In some of these colonies all the forms present—when active, and not degenerated—retain the stain when treated by Gram's method. In the first form noted in this group square or round ended motile rods were formed, which vary in length. At one end an oval or quadrangular spore is formed; the latter becomes spherical, and, breaking open, a number of microspores are let free; some of these, or bodies of a similar appearence that are present, divide by binary fission, and when mature form two bodies of crescent or half-moon shape. At this stage multiplication ceases, and parallel grouping is frequently noticed. These crescent bodies contain two triangular or pear-shaped masses, which have their bases apposed; between these a spherical body forms, and, the ends elongating and curving, a double club is formed. These clubs sometimes appear as a chain of spheres, the end ones being of larger size. At a later stage suitable staining shows that some of them form two or three oval or pear-shaped spores. The sporing variety alone has been met with in direct cultures of this fungus, but when subcultures were made the majority of the clubs present did not spore; they assume the same form as the diphtheria bacillus, and give an acid reaction in bouillon cultures, to which a trace of glucose has been added. If the solid parts of a serum culture are pressed out gently on a slide, and stained by Gram's method, these spores are seen to enlarge and contain a number of minute spherical bodies, which are actively motile in hanging-drop preparations, a bundle of flagellæ which arise from one point on their surface often giving them a medusa-like appearance. Their ultimate destination has not yet been definitely proved, but it is quite probable that they develop Gram negative properties, and are similar to the gram negative coccus-like microspores which later show polar staining.

In other colonies all the forms assumed rapidly liquefy serum, and are Gram negatives during their development to maturity. They first appear as curved or undulating rods, with round ends; they soon assume a jointed or beaded appearance, chains of endogenous spores are formed, which are identical with the bodies described by Donovan as a round cell infection in this disease. During their evolution division occurs, and chains of bodies, like enormous cocci, are formed. Microspores now appear in the medium, which undergo binary fission, and later show bipolar staining; their protoplasm now extends at the poles, and a spindle-shaped body, which contains two chromatin staining spots, results; a central constriction forms, and median division to two egg-shaped bodies occurs; some of the latter degenerate, whilst others appear to elongate and form rods, which contain a large central chromatin staining mass, which at times is placed at one end, but eventually divides, and may either be rotated to the poles, or, undergoing fragmentation, is scattered through its protoplasm. During the latter stage these bodies more than double their length, and in film preparations show a few shallow turns. They now divide into four or five spindle-shaped segments, each of which contain one or two chromatin staining granules; the ends of each segment become pointed, and, extending, threads are formed which may have rounded ends, or the latter may be drawn out to fine points. Some remain extended, and others have a twisted, spirocheta appearance. The latter organism seems to be more closely simulated if a culture of the gram positive microspores are added at this stage. Excepting the spores and microspores, all the forms assumed in this group are motile. When Donovan's bodies are forming, chains of minute rods and spheres are seen in the medium, which later on appear to assist in mycelium formation.

When the organisms of both cycles—before the stage of spore formation—were isolated in pure cultures from the skin surrounding separate lesions, and grown together till mature, clear (more or less) circular plaques appeared in the medium, the edges cleaved and split to a point near the centre, and, each extremity undergoing multiple fission, sprays of acicular rods were formed, which gave negative results when tested for fat crystals. These later form a trellis, which later is interlaced with a fine, branching mycelium, to which is attached spores and many forms of the bodies evolved during the development of both cycles. Some of these mature secondary fungi were broken up, diluted, and plated, and the spores of both cycles isolated in pure cultures. From a symbiotic culture of these, mature secondary fungi were formed in seven weeks. When a whole secondary fungus is subcultured there is generally such an enormous reproduction of the different stages that the medium is used up before maturation can take place. Further experiments have proved that when mature secondary fungi are subcultured the stages succeeding spore-division develop, and that from young fungi, which have not reached the stage of mycelium formation, the stages preceding spore-formation can be obtained.

When examining stained pus films much confusion will be avoided if it be remembered that most of the bodies engaged in these cycles are encapsuled, and that when intracellular the capsule is often unstained, the internal structure only being tinted; but when free the capsule is generally so deeply stained, and the internal structure so obscured, that they are easily mistaken for bodies of a different nature. When they have been free in pus for some time, or when surrounded by phagocytes, the capsule does not stain, and a squat, blunt, broader appearance is established, which is nearly always assumed by them in cultures.

It appears that in the tissues the early forms of these bodies have an extracellular existence, the spores alone entering the round cells; some must be extruded or destroyed, as cells are often seen filled to bursting point with them. The spores develop their respective cycles in certain limited parts of the round cell protoplasm; although they may be extruded, the microspores and their offspring remain enveloped till the later stages of their evolution are reached. When the cells are ruptured during the preparation of films, the spindle-shaped bodies, being crowded together within the enveloping membrane, naturally assume a half-moon shape. When the daily examination of the pus from one sore was continued for some months, each of the bodies evolved during the growth of both primary fungi was always seen; when the gram negative cycle was nearing completion, the fine-pointed spirochæte of Wise, or the blunt-ended ones described by Cleland, were invariably found. Excepting staphylococci, which are microscopically indistinguishable from the microspores and spirochatæ, neither of the fungi, nor the bodies produced by them in their evolution, have been seen during numerous examinations of the discharge; from open lesions of other diseases, venereal and otherwise, I have failed to find them on the pudenda of unaffected aboriginals. In active sores, in which both cycles are represented, each group always occupy separate cells, the spores do not develop simultaneously; consequently, some become matured sooner than others, two or more stages being nearly always present in each cell. This is equally noticeable in cultures, and attempts to obtain a uniform race have always failed.

Owing to lack of laboratory animals, inoculation into those available failed. Dogs, goats, and a horse were tried with negative results. Specimens were mounted from three cultures, in which mature fungi were forming. One from each tube was planted in the axillary skin of a patient who was suffering from inoperable carcinoma. In the third week one inoculation proved successful. A papule appeared, which two weeks later broke down, forming an ulcer, in which typical nodules of infected round cells were formed. The latter contained bodies of the Gram negative cycle; a few pear-shaped spores from the Gram positive club-forming stage were scattered over the films. Examination of the specimen removed from the tube from which the successful inoculation was obtained showed a barely mature secondary fungus, in which little mycelium had formed; the organisms of both cycles were present in the sporing and presporing stages. The tubes from which the unsuccessful material was taken contained fungi which were surrounded and interlaced with much mycelium, and many organisms of both cycles in advanced stages of development.

In all examinations and experiments staining was limited to Leishman's, Giemsa's, and Gram's methods. Plain bouillon and Lœffler's medium were used for artificial cultures, nutrient agar being used for plating. Cultures were incubated at 37 c.

My cases of Granuloma Pudendi, on which this paper is based, now total 106. From every patient pus films or other material was obtained, the examination of which has never failed to reveal an infection by the bodies formed during the symbiotic culture of the elements of a mature fungus.

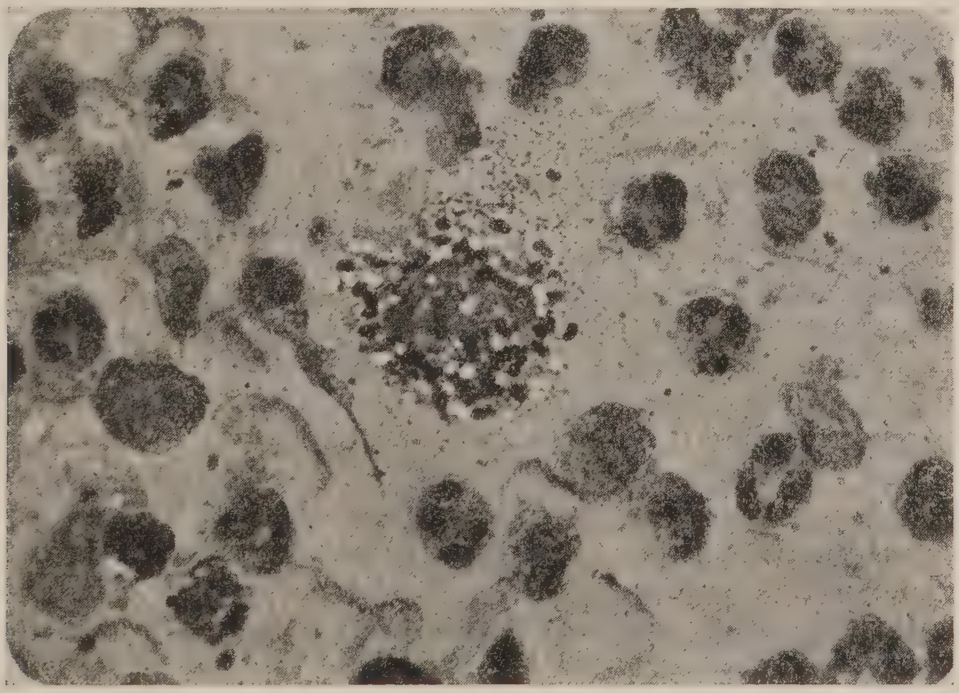
DISCUSSION ON DR. STRANGMAN'S PAPER.

Bodies like Leishmania in Films from Cases of Ulcerative Granuloma of the Pudenda.

J. B. Cleland (Sydney): Dr. C. L. Strangman, of Port Darwin, has kindly forwarded me a number of films made from pus, &c., in cases of Granuloma Pudendi in his district. An examination of these has revealed the presence of bodies which are, I think, related to Leishmania. Though some of the parasites are free, perhaps from rupture of their host cells, the majority are included in large mononuclear cells. These cells are considerably distended and the parasites often seem to lie in groups in lighter-coloured areas in their protoplasm. Large numbers may be present in one cell. Occasionally a few parasites may be seen in polymorphonuclear cells. The majority of the parasites met with in films have been very small, and their minuteness has accounted for my failure to identify them in one previous case at least. Their true nature seems more determinable, however, in some of the films forwarded by Dr. Strangman. In one set of these the bodies are numerous and large, reaching approximately 3μ x $1{\cdot}5\mu$. They were present in the large cells mentioned, or were free from rupture of these. The protoplasm of the cells containing them was vacuolated and degenerated-looking. The bodies were oval or of an elongated bean shape, and stained a purplish tint with Giemsa. When the staining was lighter and differentiation more perfect, a purplish rod-shaped body was very noticeable, bisecting the bean-shaped structure, which showed a clearer centre with purplish periphery. This periphery seemed thicker at one side and towards the ends. In other specimens the rod appeared as a purplish dot, with a purplish mass opposite to it, or with the two ends of the parasite more deeply stained.

Between these large forms found in certain films and the smallest forms which were alone found in many of the films, occasional intermediate phases were seen. These consisted of rounded to oval bodies, with one side and both ends stained, but with a gap in the other side. The minute bodies about the size of gonococci showed a similar appearance. In none of these could the rod-shaped body be identified. The minute bodies were very abundant in the cells, and resembled somewhat bipolar bacilli. They seem unquestionably, however, to gradate into the larger bodies.

Some two years ago in an article on the "Etiology of Ulcerative Granuloma of the Pudenda" (*Journal of Tropical Medicine and Hygiene*, May 15, 1909), I described in one case bodies similar to the small ones just mentioned, in films made from the lesions of granuloma of the pudenda in a white man. I described these bodies as follow: "Numerous swollen mononuclear cells with their protoplasm distended almost to bursting with large numbers of diplobacillary bodies, staining feebly by Gram's, but very distinctly by Giemsa's and Leishman's methods. These cells with their contents could be easily found in numbers in those parts of the section rich in cells. Besides these, there were numerous scattered, usually extra-cellular, bipolar bodies (not unlike plague bacilli, save that often one side as well as the two ends were stained). Their staining was poor, suggesting degenerative bacteria, which opinion was supported by Dr. Bull, of Melbourne, to whom I showed them." A photograph is also given of a cell containing these bodies.



Leishmania-like Bodies in smear from Ulcerative Granuloma

It is obvious that these bodies, which at the time were thought probably to be bacilli, were really the parasites found in this case. I had an opportunity of showing some of these original films recently to Dr. von Prowazek; he was of opinion that the bodies were probably bacteria.

In the *Lancet* (October 15, 1910, p. 1128), Dr. R. Markham Carter describes Leishman-Donovan bodies in cases of Ulcerating Granuloma of the Pudenda as met with in India. The parasites he figures seem in general to be the same as the larger ones met with by us, though I cannot make out all the details he describes, *e.g.*, the deeply staining dot near the bodies.

The examination, therefore, of Dr. Strangman's material, coupled with that from my case previously stated, and with Dr. Carter's work in India, seems to suggest a relationship of this disease to Oriental Sore. On the other hand, the spirochæte found by me in two of my original cases, and, I believe, by others since, may still be the important agent, though spirochætes are much more likely to be the contaminating factor than the present parasite.

THE OBJECT AND SCOPE OF TROPICAL MEDICINE IN AUSTRALIA.

By ANTON BREINL, M.D. (Prague).

IT is my pleasant duty to lay before you a subject which has occupied my mind for the last eighteen months—*i.e.*, the object and scope of Tropical Medicine in Australia.

Since the beginning of history the white race has moved towards the East, until races living there or coming from there prevented further migration. As the North was partly populated, or the conditions of life became too severe, migration trended towards the South. But there the climate soon seemed to form a barrier, which for a long time seemed insurmountable. A great number of the pioneers who went forth paid the heaviest toll for their undertaking by losing their lives soon after their arrival in the country of their hopes. Many parts of the tropics had acquired the evil reputation of being the white man's grave. Only within the last twenty years has science come forward, attempting to explain the cause and effect, and the progress made within this time has forced the conviction upon us that the unsuitability of the climate depends mostly on the prevalence of diseases carried by intermediary hosts, which flourish in certain parts of the tropics.

Manson's remark, that "the more we learn about these diseases the less important in its bearing on geographical distribution and as a direct pathogenic agency becomes the rôle of temperature, and the more important the influence of tropical fauna," sums up our experience.

Ross's discovery of the rôle of certain mosquitoes—the *Anophelina*—as intermediary host for bird and human malaria, followed by Read, Carroll, Lazear, and Agramonte's discovery that *Stegomyia fasciata* plays the same rôle in regard to yellow fever, and numerous other valuable results in quick succession have opened up the tropics and have shown ways and means to combat many of those diseases by practical measures, proving, for example, that malaria and yellow fever may be prevented, not only by the erection of air-tight walls, to keep away the miasma of the swamp, but by far simpler methods —the use of a mosquito-net.

The success which has followed mosquito extermination is clearly shown by comparing the death-rate of Rio and the malaria statistics of Ismailia.

In Rio—a yellow fever centre—the mortality has fallen from 3,974 in 1890 to eighty in 1905, and at present yellow fever is practically non-existent. In Ismailia, 2,284 cases of malaria occurred in 1900, and only thirty-seven cases in 1905.

The work accomplished in tropical parasitology has furthermore shown that Nature in the tropics has provided for the survival and spread of certain parasites by protecting them from the sterilising action of the tropical sun, namely, by means of encasing them within the body of an insect-carrier, the intermediary host. Whereas in densely-populated countries bacterial infections are common and the rule, in parts of the tropics only sparsely-populated the protozoan infection is of common occurrence. The latter infections are generally of a more chronic character than most of those due to bacteria, Nature providing in this way for the survival and spread of these parasites over prolonged periods, and over wide areas.

Malaria may serve as an example. Many cases are on record where people, after having contracted malaria, lived in countries where indigenous malaria is unknown, and again developed typical malaria attacks after enjoying perfect health for years.

Trypansoma gambiense, the parasite of Sleeping Sickness, may reappear in the peripheral blood and cause serious symptoms after having lain dormant in the internal organs for three or four years.

Naturally, in densely-populated parts of the tropics diseases due to bacteria and spread by contact, have made their appearance, such as plague, cholera, and dysentery.

The object of tropical medicine involves much more than the study of parasites and diseases occurring in the tropics; it comprises in its working sphere the welfare and life of the white man under new, and, to him, artificial conditions.

The white race, born and bred for numbers of generations under the northern skies, in countries which enjoy a shorter or longer cold spell every year, has migrated to regions where the temperature is charged with humidity and the tonic of a cold winter is lacking. These countries are extremely valuable to the race on account of their natural wealth, and the simplicity of life found therein, Nature providing food throughout the year without a considerable amount of labour, all of which conditions tend to have a debilitating and enervating effect upon the newcomer.

Moreover, their organisms had become accustomed, throughout many generations, to certain diseases and specific infections prevalent in temperate climates, and had acquired a relative impermeability of their living matter for certain infections, and infectious disease being produced only by the disturbance of this impermeability.

On transplanting the white race to the tropics this acquired impermeability has become upset. The organism has become exposed to the invasion of infections to which it has hitherto not been accustomed.

Parasites, against which a relative impermeability has been acquired by native races in whom they act as parabiontes—the invading micro-organisms receiving advantages, and the infected organisms not suffering any disadvantages—become true parasites in the newcomer, invading his organisms without finding any resistance whatsoever, and live at his expense.

Take the negro races in Africa, who often become infected in childhood with malaria, and may carry the malaria parasite in their system during the greater part of their life without showing any clinical symptoms. Take the internal parasites which abound in the intestines of the healthy aboriginals of the tropics, but which, when invading the newcomer, give rise to serious diseases. Consider the animals living in the tropics, as birds, reptiles, and mammals, which are swarming with intestinal and blood parasites, without being the worse for their presence.

Adaptation, taken in this sense resolves itself into the question how the organisms exposed to the invasion of certain micro-organisms for which they had no impermeability could overcome this invasion without detrimental effect.

A few years of experience forced the conclusion on the white pioneer that there is only one way of acquiring this impermeability and immunity, and this is by avoiding the infection. But this can only be accomplished through the careful study of local conditions and diseases, the mode of spread of these diseases, and the extermination of the infecting organisms. And as health is the first postulate to prosperity for the State and for the individual, this conclusion has given rise to the establishment of a new branch of medicine—tropical medicine—dealing as much with the investigation of existing diseases as with the finding of ways and means to prevent their introduction and their further spread.

In future the pioneer should not be the settler, but should be the scientifically-trained man, who studies local conditions in a new territory, and investigates the existing diseases, and the possibility of the introduction of other diseases, devising at the same time ways and means how to avoid the infection, and requires therefore, as Ross points out, " a knowledge not only of biological technique, but of the broad principles of statesmanship."

In the foregoing sense, the object of the Australian Institute of Tropical Medicine is: Firstly, the careful consideration of the question how far the insular isolation which Australia has enjoyed up to comparatively recent times with regard to diseases will protect Australia in the future; secondly, the study of existing diseases in tropical Australia, and their prevention; thirdly, a thorough and impartial inquiry into the physiology of the white race living and working under different conditions in tropical Australia.

Northern Australia is surrounded by numerous islands, some of which are densely populated, and are hotbeds of more or less deadly diseases, which diseases, on account of the increased and quicker traffic, can hardly be kept out for ever by quarantine regulations alone.

The Dutch East Indies are in close proximity to Australia, and smallpox has been introduced in past years, and has played havoc amongst some of the aboriginal tribes living on the North Coast.

There is China, with its numerous intestinal parasites, its dysentery and occasional outbreaks of plague. There is New Guinea—part of it, from a medical point of view, unexplored, and perhaps unexplorable—with its malignant type of malarial fever and its dysentery.

The malignant type of malaria has already been introduced into the Northern Territory by miners. During the last two years dysentery has made its appearance in Thursday Island, after having worked its fatal course along the Torres Straits Islands, stopping short there on account of the lack of population between Thursday Island and Cooktown.

Much has been said of late about the possibility of the introduction of yellow fever into Australia. Up to the present yellow fever has not made its appearance in the East, as ships from infected regions had to pass through a cold climate before reaching their destination. The opening of the Panama Canal offers a straight run for ships through tropical seas from Central America, where yellow fever is endemic, to the East and Australia.

It is a well-known fact that Stegomyia fasciata may travel for long distances on ships, and if infected, may give rise to a serious outbreak, the more serious for Australia as this mosquito occurs in great numbers all along the Queensland coast, where in the summer-time the temperature is high enough to favour the development of the yellow fever parasite in the intermediary host, and cause the disease to spread.

That mosquito-borne diseases may be introduced by means of ships is possible if it is considered that ships are equivalent to infected houses as regards conditions as to food, water, and temperature.

Only in 1909 a yellow fever outbreak occurred in St. Nazaire, on the south coast of France, due to infected stegomyia taken on board ship in the West Indies, but, like all previous outbreaks in countries where stegomyia is non-existent, was limited to those who came in contact with the ship, and were exposed to the bites of the infected mosquitoes.

Fumigation of the ship does not kill the mosquito unless it be done very thoroughly. Moreover, as the diagnosis of yellow fever is very difficult, and in many cases in the early stages almost impossible, a serious epidemic therefore would arise if infected stegomyia be once introduced; unless tropical Australia follows the example set in other parts of the world, to exterminate as far as possible the mosquito, which, on the whole, is a simple matter.

It would seem advisable to study the diseases prevalent amongst the aboriginal inhabitants of the lesser known islands north of Australia, who occasionally visit the mainland, and are likely to leave behind mementos which Australia may not forget for some time.

The study of diseases prevalent in tropical Australia furnishes a plentiful supply of material for the Institute.

I may point out some of the work which promises valuable and practical results.

Dengue Fever is prevalent in North Queensland. More or less widespread epidemics occur every year in different parts. The fever attacks the victim very suddenly, causing intense pains in the back and bones, which symptom gave it the popular name of Break-bone Fever. After a few days of continuous fever a general rash makes its appearance, resembling somewhat the scarlet fever rash. Soon afterwards the temperature subsides, giving place to a well-marked nervous depression, from which the patient only very slowly recovers. The parasite is not known yet. A few experiments only tend to prove that these parasites are spread by the bite of our common house-mosquito—Culex fatigans—which abound all over the north of Queensland. Its micro-organism passes through a Berkefeld filter, which does not allow the micrococcus Melitensis to pass.

Dengue fever is not a fatal disease; but considering that sometimes epidemics sweep over whole towns, incapacitating the victims for some time from work, it is an economically important disease, causing a good deal of loss in money and labour.

Besides dengue fever, there are *local fevers* in different parts of tropical Queensland, at present classed under different popular names, awaiting elucidation concerning their etiology.

The distribution of *malaria* in Queensland has not been mapped out. Malaria fever has certainly ceased to exist in larger towns, but cases occur sporadically in many parts of Northern Queensland, especially in scrub country. In the Northern Territory malaria is very prevalent, and during our recent journey we have been able to find, after leaving Pine Creek, the parasite of benign and malignant tertian malaria in some of the patients examined, and nearly everybody we met had had fever in the past.

It is interesting to note that our findings point to the fact that malignant tertian fever has been introduced by miners from New Guinea, who, on leaving this country, harbouring the Gametes in their blood, set up epidemics of varying severity in the new localities they choose for their field of action.

Filariasis is of frequent occurrence in North Queensland, and is a disease which offers a good opportunity for original investigation. In some cases Filariasis is not a serious complaint, as a good many people may harbour the parasite in their blood without showing any clinical sign of the infection. But numerous cases come under observation where the presence of filaria gives rise to serious symptoms. The presence of adult filaria in the lymph system causes soft swellings in the groin, sometimes quite insignificant, but sometimes as large as a man's fist. Frequent attacks of orchitis cause the testicles to swell up to five or six times their original size, and give the patient a great deal of pain and discomfort. These changes are accompanied by more or less frequent attacks of fever. Lymphangitis, leading to abscess formations, may occur in any part of the body, on the arms, legs, scrotum, or on the abdomen, which abscesses are extremely painful, and when opened up discharge a thick sterile pus.

Chyluria is not unfrequently found, the patient passing a thick creamy-coloured urine, which coagulates into a solid gelatinous mass soon after it is passed. A frequent complaint is that of a dull pain in the back, in the lumbar region, which prevents the patient from following his occupation.

Elephantiasis is, in all probability, due to a filaria infection, and has been met with in Northern Queensland.

The diagnosis of filariasis is fairly simple in most of the cases, as the microscopic examination of the peripheral blood at night-time reveals the presence of microfilaria in smaller or larger numbers. In the daytime the embryos usually recede from the peripheral circulation to the internal organs, and a great number may be found in the lungs.

How is the infection contracted? In the year 1879 Manson proved that Microfilaria Bancrofti undergoes further development in the mosquito—Culex fatigans—commonly found in great numbers all over the tropics. Other mosquitoes—as different Anophelina, and Mansonia uniformis in British Central Africa—may act as carriers.

Manson was able to follow the development of the microfilaria after they had been taken up by the mosquito with the blood. The young microfilaria lose their sheaths in the stomach of the mosquito, penetrate the stomach walls, and make their way to the thorax muscle of the mosquito, where they rest between the muscle fibres, undergoing further development. After a varying period, dependent on temperature and other conditions, mature embryos—usually a pair of male and female—reach the labium and enter the new host, escaping through the rupture of a fine membrane stretched out between the labellæ and the concave tip of the labium.

The microfilaria, after entering the new host, reach the lymph system, and develop into the adult worm, the female being about 8 to 9 cm. long, the male only about 4 cm.

In Queensland filariasis is of fairly common occurrence. It has been found in Brisbane, where about 10 per cent. of the hospital population were found to be infected.

Numerous cases came under our notice who had contracted their infection in Townsville, others in Charters Towers, Winton, Port Douglas, Normanton; and two New Guinea natives, examined at Thursday Island Hospital, harboured the microfilaria in their blood.

As Culex fatigans is found in great numbers in the towns of Queensland, and Mansonia uniformis in great numbers in the bush in different parts, it seems strange that there are comparatively so few cases. As the mosquito is all-prevailing, and cases of filaria scattered all through the townships, hardly anybody could escape the infection were the transmission a simple and direct transmission as in the case of malaria.

The study of the life-history of this parasite in relation to the different species of mosquitoes, the fate of the embryo after entering the human body, and its migration of the blood-stream into the lymph-system and its fate there, require further elucidation.

An allied species, filaria immitis in dogs, is of frequent occurrence in Townsville, and a close and careful study of this parasite may throw considerable light on these questions.

The treatment of filariasis is, up to now, only symptomatic. The discovery of a treatment which would attack the adult worm would be of great value. All our attempts in this direction have not been followed by success.

Of intestinal affections, *Sprue* is not of rare occurrence, and cases which had contracted this disease in the Innisfail district have been seen.

Sprue is a chronic catarrhal inflammation of the mucous membrane of the intestinal tract, with alternate improvements and aggravations of the condition, leading to an intestinal atrophy of the mucous membrane of the tongue and the intestines.

The first symptom, as a rule, is the appearance of small raised red spots on the edge of the tip of the tongue, which very soon loses its natural roughness and becomes smooth, red, and dry. The liver atrophies, and the patients, who emaciate to a great extent, show a dirty, yellow complexion. Characteristic is the passing of numerous thin clay-coloured frothy stools.

The etiology of Sprue, as well as the mode of infection, is not known.

Dysentery does not occur to any extent in Queensland; sporadic cases may be observed now and again. During the short time of my stay in Queensland two epidemics have been observed in Thursday Island, affecting mostly the natives of the pearling boats. Cases also occur amongst the white population.

Both epidemics were introduced from New Guinea, where dysentery is endemic, and where epidemics of greater or less extent occur at intervals.

The question of the etiology of dysentery in New Guinea has not been studied up till now, and results obtained in this direction would certainly be of importance, as New Guinea is so near to Queensland, being connected by a continuous chain of islands with the mainland, which afford every facility for an epidemic to spread to Queensland.

Ankylostomiasis is fairly prevalent in some parts of Northern Queensland, and the study and eradication of this disease would be of the utmost practical importance.

The etiology of *Ulcerative Granuloma*, a disease mostly confined to the natives, but attacking the white people now and again, is not known up to the present.

There occur, among the aboriginals in the Northern Territory, diseases which have not yet been classified. I only mention one disease, the natives suffering from which it emaciates to a great extent, resembling skeletons. This disease has been attributed to the presence of the tubercle bacillus. One case which came under my notice did not show any characteristic lung symptoms, but the liver and spleen were considerably enlarged. Blood-films of this case were examined, but did not show any parasites.

From our experience in other tropical countries it would seem imperative to study all the diseases endemic amongst the aboriginals, as on closer settlement these diseases may spread amongst the white settlers, and may cause great havoc and mortality, which could be prevented if proper precautions were taken in time.

Besides the diseases mentioned, there are a good many less-known diseases well worth a thorough study.

There are skin diseases, such as *Barcoo Rot*, a disease which manifests itself in the form of extensive superficial sores on different parts of the body, showing a tendency to heal up, but break out again.

An eye disease, *Trachoma*, occurs in Western Queensland, but as far as my experience goes it has not been decided yet whether this is analagous to Trachoma occurring in Europe.

Comparatively little is known about the intestinal parasite.

The *third object* of the Australian Institute of Tropical Medicine, and not the least important one, is a thorough and impartial inquiry into the physiology of the white race living and working under different conditions in tropical Australia. Suggestions of work in this direction had been brought before the Prime Minister by a deputation of members of the last Congress of the Australian Medical Association.

A great deal of work on the question whether the tropics in general were suitable for colonisation by the white race has been done, and is being done, but the results have been contradictory, and in a good many ways unsatisfactory.

Some scientists, who had a great deal of experience on tropical life, upheld the opinion that the tropics are as healthy for the white races as a temperate climate, provided the necessary hygienic precautions are taken and a suitable life led. Other investigators, however, maintain that tropical countries are unsuitable for colonisation by the white race, and degeneration could only be avoided by the white man taking up the habits and customs of the aboriginal races—in other words, depriving himself of culture and education.

The first generation, in their opinion, may withstand the influence of the tropical climate, but in the second generation signs of degeneration are supposed to make their appearance. If their opinion is correct, is it the tropical climate alone which causes degeneration? Is it due to the surroundings and influence of black servants and the lack of higher education, or is disease alone the *conditio sine quâ non* for the supposed inferiority of the white race living under the tropical sun. Tropical Australia should be an ideal locality to decide this question definitely, as it is a country where comparatively few diseases are prevalent, and where the aboriginal races are practically dying out. But the question of the suitability of the white race for the permanent colonisation of tropical Australia and the tropics in general is a very complex one, and cannot be decided by a few months' work, but would have to extend over a good many years. One would have to prove first that people living a life-time in tropical Australia have degenerated. As a nation consists of individuals, one would have to consider, in order to come to a definite conclusion, the individual as such; and secondly, in his relation to his fellow-citizens.

But more important results would be obtained if the second and third generations were examined. A second generation is being raised at present. A true third generation is comparatively rare, on account of the recent settlement of Northern Australia.

According to the individual view of the investigator it would be possible, by means of a few anthropometric measurements of school children, to decide the question of presence or absence of signs of degeneration in either way. To be of any practical value, a great number of anthropometric measurements of all school children in the north would have to be taken, and would have to be considered in accordance with the locality.

North Queensland is a country of diverse climates. It comprises coastal towns with a humid, hot summer and a fairly cool winter; and the high tablelands, only a short distance away, with a temperature which enables the growing of English fruit, and possessing thermometer readings below freezing point in the mornings of the winter.

My short and limited experience does not enable me to pronounce any definite opinion on the suitability of Northern Queensland and the Northern Territory as a white man's country. But comparing it with other tropical countries, it seems far superior, as far as climate is concerned; and, given the hard-working, sober settler, there should be no difficulty whatever in successfully populating the north of Australia.

So much is being said about the "thin" blood of the northerner that systematic blood-counts were made, especially on pale-looking individuals, but in most instances our ordinary examination methods showed no appreciable difference from the normal as regards hæmoglobin content and number of formed elements. The pallor of the skin was, in these cases, only skin-deep.

Important work could be done, taking into consideration not only the pathology, but also the physiology, of the human organisms under tropical conditions. Hardly any observations in this respect have been recorded.

Is the body temperature the same as in Europe? Does the internal metabolism occur in the same way as in a temperate climate? Is the blood pressure raised? Our tentative work in this respect has shown that there is certainly a slight rise of body temperature during the hot hours of the day, when the temperature rises, after exercise, about ·7 to 1 degree. It is not likely that this temperature rise involves a marked change in the oxidation process of the organisms. Few experiments have shown us that the blood-pressure in apparently normal individuals is raised to a certain, sometimes even a considerable, extent. It would be interesting to investigate how far outer circumstances influence the organic functions of the white man in the tropics.

Any results obtained in this direction may give us a valuable clue, and prove that the unsuitability of the Northern Australian climate for certain people is not due to climate, but to their own mode of living.

In the discussion which followed, Drs. Nisbet and MacDonald, Professor Anderson Stuart, Mr. O. C. Beale, and the President (Dr. Love) took part.

The following resolution, moved by Dr. Nisbet, and seconded by Professor Anderson Stuart, was carried unanimously, viz.:—"That it be a recommendation to Congress that representations be made to the Federal Government, with a view to securing for the Australian Institute of Tropical Medicine such increases of funds and staff as will enable an organised inquiry to be undertaken without delay into the various matters likely to affect the permanent establishment of a working white race in Tropical Australia."

The further resolution, moved by Dr. Elkington, and seconded by Dr. Ireland, was carried unanimously, viz.:—"That it be a recommendation to Congress that the permanent settlement of a working white race in tropical Australia be adopted as the principal subject for discussion at the Brisbane Congress when held."

DISCUSSION ON DR. BREINL'S PAPER ON THE SCOPE OF THE AUSTRALIAN INSTITUTE OF TROPICAL MEDICINE.

Dr. Nisbet, Townsville: With regard to the important question of the settlement of white races in the tropics, the general opinion seems to be favourable to their permanent and successful establishment. That it is an experiment, however, is also admitted, as the march of events in the world's history has not yet allowed such an attempt being made hitherto on the same exclusive and extensive scale as we are proposing to do in Tropical Australia.

Against this favourable view, and one which Dr. Breinl appears to share, I wish to raise a protest, and speak with no uncertain voice, basing my views on experience gained through residence in North Queensland for nearly a quarter of a century. I am not alone in taking this pessimistic view of a question which is of immense social and political importance in the future history of our country.

Lord Dudley, the late Governor-General, whose position would not allow him lightly to touch on any controversial matter, on a visit to Townsville a few weeks before his departure from Australia, in a public speech made the statement that "the question of labour being entirely confined to Europeans was one which would probably have to be reconsidered."

At the Inaugural Address of this Congress, Dr. Pockley also allowed himself to make an important reference to this subject.

Now, under the prominent circumstances that this utterance was made, it could only be the result of well-matured judgment, and a firm conviction that it would be open to criticism, and could be defended.

Dr. Breinl has probably included all the important factors bearing on this matter, when he says, "Is it climate or disease which affects Europeans in the tropics"?; and those who have interested themselves in it will have noticed how an attempt is being made, small at present, but marked by increasing efforts to try and first eliminate the special diseases which affect residents in the tropics, and when that has been done, and that factor has either been successfully got rid of, or reduced to unimportant dimensions, there would be time enough to see if the other force at work, viz., climate alone, was going to seriously affect the residents in Northern Australia, and render them unfit to continue in occupation of the country as permanent dwellers and workers.

Such a method of attacking this important question is likely to be an expensive and disastrous one if the final result turns out to be unsatisfactory, as I maintain it will, and our race is found in future generations dwindling away to a few highly neurotic, anæmic people, incapable of reproducing their species, unless fed with a constant stream of European immigration.

Now, many things may affect the influx of Europeans into Australia—war and pestilence among others—and we have no right to rely on this element to keep up the stamina of future generations.

Whether we shall ever succeed in stamping out the protozoal and parasitic diseases which are peculiar to these districts, is an uncertain question—it is a huge task, and all the forces of Nature are ranged in combat against us.

Admitting that we do, I still maintain that climate alone is sufficient to oppose our efforts at successful colonisation.

I may refer to the districts of West Africa and Panama, where medical research has achieved marvellous results in stamping out the diseases indigenous to those climates. But would any of us now select to remove ourselves to either of those places with our families to make a permanent home, and expect to flourish?

Without attempting to elaborate all the factors arising from climatic influences which may be at work in bringing this about, I would briefly mention a few which, as the result of my experience, I consider are important indications of race degeneracy which has already set in, and will become more marked through succeeding generations of native-born.

It is useless to say that their very appearance does not stamp tropical residents as not presenting all the characteristics of healthy human beings. One has only to travel between North Queensland and, say, Sydney or Tasmania, to notice the healthy robust colour and appearance of the latter, and the absence of it in the former.

You may say that blood deterioration, loss of weight, and a muddy complexion are essential to health in the tropics. I doubt it. Anyway, we would prefer, if only on æsthetic grounds, that those about us should continue to present those admirable personal characteristics which are one of the joys of life. The women are deteriorating rapidly, and handsome women, and pretty healthy children, are becoming a scarce element in our northern community. That may be a matter of opinion only, and I submit it for what it may be worth; but my medical experience has brought me face to face with hard, undeniable facts which I maintain go to prove my case.

I allude to lack of mammary development among the women, rendering far too large a number unable to nurse their offspring, which must interfere with the survival of future generations.

The enormous number of abortions among the married women, apart altogether from these criminally induced, shows a weakening of the reproductive function, which will lead to diminishing birthrate, if not to total race extermination.

The third piece of evidence is the abnormally large number of convulsive seizures in young children, from slight causes, and from no ascertainable way. Whole families are affected in this way, and though permanent nerve diseases may not follow in this generation, succeeding ones must suffer from the unstable nervous system which is rapidly arising.

This is a most important question, and one which ought to be made a prominent, in fact, a leading feature in a succeeding Congress. When the meeting takes place in Brisbane, a suitable opportunity to discuss this question from every standpoint will present itself; and in order to give an opportunity to collect material and conduct investigations which can be submitted to that Congress, I consider the work of the Tropical Institute in Townsville should be largely increased, and an efficient staff appointed to assist the Director in carrying out this important work.

Dr. W. C. C. MACDONALD: Mr. President and Gentlemen,—I have heard with pleasure and beg to congratulate Dr. Breinl on his extremely interesting sketch of the chief tropical diseases prevalent in North Queensland.

It is impossible to overestimate the importance to the community of the establishment of a bureau in Townsville for research into the diseases peculiar to the tropics, and which have given it the unenviable, and, in my opinion, most undeserved, reputation as a place unfit for the white races to live in.

From the remarks of Dr. Breinl, and from the comprehensiveness of his programme of procedure, and from his undoubted fitness for the task, we may venture to predict that the tropics will at last, and in the near future, receive at least justice. The climate of the tropics has long enough had to bear the odium which should properly belong to the diseases with which it is affected, and I for one am a firm believer that the more this matter is scientifically investigated the clearer will become this great truth; and I venture the prophecy that at no very distant period of time, when science has solved the problem of effectively combating these diseases, white races will flock to the tropics, and there realise materially the dream of ages, an Arcadian existence.

With regard to Dr. Nisbet's remarks, I may claim, having resided on the coast of North Queensland in the latitude of " the Gulf "—the embodiment of all that is supposed to be unhealthy—for a period of twenty-seven years, some acquaintance with the subject, and I am glad to say my experience leads me to totally disagree with the pessimistic view that we have heard from Dr. Nisbet. I have seen as fine children—not only a few, but numbers—born and reared in North Queensland as could be found in any part of the world; but about 70 per cent. of the children suffer more or less from that fell disease, Ankylostomiasis, which, when untreated, has undoubtedly a most deleterious effect, both physically and mentally, on children. But fortunately this disease is one for which science has already pointed out effective treatment, and is capable of being exterminated. The women who suffer in health from residence in the north of Queensland are, for the most part, sun-haters, and never, if they can avoid it, expose themselves to the direct rays of the sun, and undoubtedly the sun's rays are as essential to nutrition in the human species as to plants. I think that direct sunlight as a life-giving and stimulating factor is not sufficiently appreciated. Women of the working classes in the tropics, whose complexion is more like that of men, enjoy as good health as the average woman elsewhere.

The whole question of the fitness or otherwise of the tropics as a place of abode of white races is an intensely interesting one, and one of vital importance to Australia, and can only be solved by unbiassed investigation by men of experience, fitted for the task.

ANKYLOSTOMIASIS.

By Anton Breinl, M.D. (Prague).

The subject chosen for to-day's discussion is of great practical and economic importance to North Queensland. Ankylostomiasis is a disease fairly prevalent in the coastal towns of North Queensland, and plays havoc among the infected children and adults. That it does is the more pitiful, as Ankylomostiasis is a disease which can be easily prevented and eradicated if proper precautions and measures were taken.

Ankylomostiasis is not confined to the tropical and subtropical countries only, but occurs also in temperate climates under special conditions—*i.e.*, in localities where the temperature is fairly high, and even during the whole year, as in deep coal-mines. It is prevalent in the southern parts of the United States, on the east and west coasts of tropical America, in Italy, Africa, India, China, and in the different islands, as Java, Sumatra, Philippine Islands. The disease has been prevalent in the coal-mines of Belgium, England, and Austria, and some of the deep coal-mines in Westfalia were at one time hot-beds of this infection.

The parasite Ankylostoma duodenale was discovered by Dubini in Milan in the year 1845, and since then has been found frequently, the more often the more carefully it was looked for. The small worm which causes the disease belongs to the large family of the Strongylidæ. The male is about 10 cm. long, the female about 12 to 13 cm. The worm has an ovoid characteristic mouth, lined with a chitinous membrane, carrying ventrally two pairs of hook-like teeth; in the floor of the mouth-capsules two ventral chitinous blades are present. The Cursa in the male is umbrella-shaped, and is supported by chitinous rays, which are arranged in a characteristic manner. Through the opening of this Cursa two spicules, fine thread-like hairs are projecting. The ova are oval, with broad round poles, the egg-shells being always separated by a considerable space from the cells. The genital opening of the female opens out about two-thirds of the length. During copulation, which takes considerable time, both worms are in a rightangular position; the male fixes its caudal bursa over the vulva of the female and introduces the spicules into the vagina.

Life History.

The adult worms live in the duodenum and the jejunum of man, where the females lay a considerable number of ova, which are passed out with the fæcal matter, as a rule in the four-celled stage. The further development of the ova takes place outside of the human body, and depends on three factors—on humidity, high temperature, and the presence of oxygen.

The cells of the egg continue to divide for about twenty-four hours, and after this time, if the conditions were favourable, the embryo is found coiled up within the egg-shell. Then the egg-shell splits on one end, and the little embryo escapes, moving about slowly, feeding on fæcal material. The optimum temperature for its development lies between 25 and 30 degrees; a temperature of 50 degrees, or freezing point, kills the egg. The ova in the surface layer of the fæcal matter always develop more quickly than the deeper-seated ones. Sunlight tends to kill the embryo, diffused light retards development, and darkness favours the development. During the early stages the mouth of the embryo is characteristic of the species, being fairly long and cylindrical. The œsophagus is long, ending bulb-like. After three to five days the first ecdysis takes place, without alteration in the form or shape of the parasite. After varying intervals the form of the embryo changes, it becoming larger and losing its characteristic-shaped mouth, and the second ecdysis is prepared. The old skin becomes detached from the body —is not shed, but surrounds the embryo in the form of a sheath overlapping the anterior and posterior ends, so that the embryo can move to and fro within the sheath. At this stage the embryo ceases to grow, and the mature larva contains a certain amount of reserve material in the intestine in the form of dark-coloured granules. The embryos now leave the fæces, and attempt to reach water or damp soil, where they may rest for a month unchanged. In stagnant water the larvæ sink to the bottom. If the temperature is favourable, the larvæ come up to the surface and climb up suitable objects, waiting for an opportunity to infect man.

Eggs and immature larvæ swallowed are not infectious; mature larvæ, however, taken up with the water give rise to an infection. On reaching the intestines of men the larvæ undergo another ecdysis, forming a round mouth capsule with four small teeth situated cross-wise. They fix themselves by means of these teeth to the epithelial cells of the gut-wall, and after the fourth ecdysis assume their definite shape. The genital system develops, and after about eight days the first copulation takes place. About four to five weeks later the eggs are numerous enough to be easily detected in the fæces. Infection by mouth takes place occasionally, but is not the rule.

Looss was able to prove that the parasite usually enters the host through the skin. After reaching the surface of the skin of man the embryo sheds its sheath and enters through the hair follicles, making its way through the subcutaneous tissues, obtaining access to the blood and the lymph stream, reaching in time the heart and lungs. If the number of embryo which enter the skin is only small hardly any signs of irritation are noticed; if, on the other hand, the number is fairly large the skin becomes inflamed, and a burning and itching sensation is felt. In this case the skin swells up, and a local œdema is seen. Finally, the embryo reaches the intestine, after having made its way by the way of the trachea, œsophagus, and stomach. Once the intestine is reached the embryos fix themselves to the mucous membrane of the intestinal wall. The larvæ which enter the lymph vessel make their way to the right heart, meeting there the larvæ which had obtained access to the blood stream. The larvæ have to pass the lymph glands, usually in the groin or in the axilla, and there they can be found sometimes in large numbers.

The question of the mode of feeding of the worms has been a much disputed question for a long time. Looss has been able to show on cross section that Ankylostoma duodenale does not feed on blood, as originally supposed, but that the worm feeds on the mucous membrane of the intestines. On suitable sections of worms fixed to the intestinal wall the mucous and submucous membrane forms a kind of a prop within the mouth capsules of the worm.

One of the most pronounced clinical symptom of Ankylomostiasis is the pallor of the skin, which has given rise to various names—as miner's anæmia, Egyptian chlorosis, &c., an anæmia which is due to a marked decrease in the hæmoglobin content of the blood and a decrease in the number of the red blood corpuscles. The total volume of the blood is said to be increased. The mechanism of the production of this anæmia has not been cleared up satisfactorily. The anæmia cannot be due to the loss of blood alone, but is in all probability due to the production of a toxin in the metabolism of the worm. Smith and Kilborne's finding of a substance in the anterior part of Ankylostoma caninum, which inhibits the coagulation of the blood, makes it additional evidence. Daniels' finding of a yellowish pigment in the liver and spleen cells, giving hæmatoidin reaction, supports this hypothesis.

We have injected extracts of ankylostoma into rabbits and guinea-pigs, but have not been able to produce any marked blood changes.

A worm similar to Ankylostoma duodenale, but morphologically different, producing the same symptoms, has been found in the Southern States of the United States of America, in Brazil, the Philippines, India, and Africa. It differs from it in many respects, the neck being more slender, the mouth capsules having, instead of the ventral hook-like teeth, two small blades and one pair of dorsal conical teeth.

Similar worms to Ankylostoma duodenale are found in different animals, having been seen frequently in the intestines of cats and dogs in Townsville. This worm, Dochmius trigonocephalus (Ankylostoma caninum), possesses three pairs of teeth on the ventral surface of the oral cavity. This parasite sometimes causes fatal epidemics amongst cats.

Ankylostoma duodenale is pathogenic for men, and produces, according to the number of worms present, a more or less severe disease. In most cases a marked and progressive anæmia makes its appearance, the conjunctivæ becoming pale and colourless. Most of the clinical symptoms are secondary to the anæmia. The patients complain of a feeling of tiredness, being unable to work, and the least exertion causes palpitation of the heart; some cases are intensely emaciated, other cases show a more or less marked œdema. The symptoms, at first sight, could be mistaken for those of nephritis, were it not for the profound anæmia.

I have seen cases where the eyes were completely closed up on account of the heavy œdema of the eyelids, where the limbs were twice the normal size, the patient being unable to move or sit up in bed.

Patients suffering from Ankylostomiasis complain frequently of a dull pain in the region of the duodenum. Infected children always show more or less severe symptoms; in fact, they can easily be picked out at a glance. They are pale, always show more or less marked œdematous swelling, they are mentally dull, and do not join in the play of their companions. By the parents they are always described as obstinate children, who break out in tears at the least provocation, and cannot be ruled. In chronic cases, and especially if the infection extends over a long period, the symptoms are very severe, and these children are markedly backward in their development.

One case was brought to the hospital of a neglected child about 5 years of age. The child could not walk, did not speak, but lay in its bed listless all day long. After a severe treatment extending over nearly four months the child had quite changed, and had become a favourite with the nurses and patients.

Much has been said about the habit of infected children eating earth, a symptom which has given this complaint the name of the Earth-eating Disease. Not only are sand and earth swallowed by these children, but very often their perverted appetite manifests itself in eating paper, pencils, wood, and other indigestible articles. In all probability this perverted appetite is the outcome of the intense intestinal irritation caused by the worms.

The diagnosis of Ankylostomiasis is, on the whole, simple in fresh cases, as the characteristic eggs are usually found in large numbers in the fæces. In chronic cases the diagnosis may be difficult, as the number of eggs passed is often very small, and even the examination of eight to ten coverslip preparations may not reveal their presence. But even in these cases the eggs can be found after centrifugalising an emulsion of fæces or keeping a certain amount of fæcal matter for some time at a temperature of 28 to 30 degrees centigrade, when the free embryos usually work their way to the surface of the infected matter.

One symptom which has been pronounced as characteristic of this disease is the relative increase in the number of the eosinophile white blood cells, but as eosinophilia occurs also in other diseases, as filariasis, and in infections with other intestinal worms besides Ankylostoma duodenale, it cannot be relied on. The diagnosis can only be verified by the finding of the ova in the stool.

The treament of Ankylostomiasis is, on the whole, very simple; in some cases, however, offering great difficulties. We are fortunate enough to possess two specific remedies, Thymol and Betanaphthol which are known to be potent vermifuges for Ankylostoma duodenale, but unless the treatment is done carefully, and with the proper precautions, it is either useless or even dangerous. For successful treatment, it is necessary to keep in mind the mode of action of these drugs. The parasites are not killed in the intestines, but only stunned by the drug, causing them to loosen their hold of the mucous membrane, and may be removed afterwards mechanically through the administration of strong laxatives. It is therefore necessary to clear out the intestines well before the administration of vermifuge by means of strong laxatives, so as to prevent the drug becoming too much diluted in the intestine with fæcal matter. Which drug is preferable: Thymol or Betanaphthol? In cases where one is able to clear out the intestines for a day or two beforehand, Betanaphthol is preferable, but only because it is easier taken and does not possess the nauseous smell and taste of Thymol. Should this be practically impossible, Thymol is preferred. Our routine treatment, therefore, consists in the administration of a laxative for twenty-four hours before the vermifuge is given, usually calomel and jalap in large doses. The patient is starved for about eighteen hours before the drug is given. Early in the morning we administer Thymol or Betanaphthol.

With regard to the dose, we give as much as 120 grains of either in four or five half-hourly doses to an adult, and follow this up again by a laxative. One has always to keep in mind that Thymol is a poison, and may, if absorbed, give rise to severe toxic symptoms, and even death. Thymol is nearly insoluble in water, but easily soluble in alcohol, fat, and oils. For one day after dosing with Thymol the patient ought not to take any food containing fat or alcohol.

We usually administer Thymol and Betanaphtol in gelatine capsules. The results of treatment in this way are better than if only administered in cachets. Three to four successive administrations of Thymol and Betanaphthol, alternating in weekly intervals, have given the best results.

To give small doses of Thymol is worse than useless, and in our experience cases treated in this way are most difficult to cure, as it seems that the worms may acquire an immunity. Sometimes these cases are incurable, and we have had more than one case where the most rigorous treatment was unable to bring forth amelioration of the symptoms. In these cases the only drug which seems to be effective is eucalyptus oil and chloroform—about 40 minims of eucalyptus oil, 1 drachm of chloroform, and 1 oz. of castor oil.

Much has been said about the prevalence of ankylostomiasis in Queensland.

It is certain that ankylostomiasis shows a fairly wide distribution. We have seen cases from Proserpine; we have seen fairly numerous cases in Townsville, especially in children living in the low-lying sandy localities of South Townsville. We have seen numerous cases from Innisfail, Cardwell, and from Cairns, but the district between Cardwell and Cairns seems to show the widest extent of the infection. Especially of late the disease seems to have spread amongst the workers in the sugar-cane fields of the Innisfail district, where I treated, on one occasion, three farmers from adjoining selections, all being infected with this parasite.

Nearly every case which was treated in the hospital had suffered some time ago from a skin lesion on the foot. It had appeared in the form of an inflammation of the skin, the seat of predilection being the instep. The skin first becomes of a reddish colour, then the inflammation soon begins to spread in spirals, the patient complaining of a creeping and burning sensation. Whether this peculiar skin inflammation is due to the entering of Ankylostoma larvæ or to some other parasite can only be decided after careful observation.

I have noticed a similar skin lesion occurring in Cairns. In one home I visited I found every inmate affected with this peculiar lesion. Unfortunately, I have not been able to follow the later history of this family.

It seems a very serious matter to have Ankylostomiasis even only present in country districts of Northern Queensland, where there is ample opportunity for the spread of this infection—as, for example, Innisfail, with its enormous rainfall and its sandy soil.

At present it would be an easy matter to treat the infected patients and advise modes and means of eradicating the infection. But I am certain that in time, when the infection has become further diffused, and when large areas have become infected, preventive measures will be far more difficult and costly.

In Queensland Ankylostomiasis is fairly widespread amongst young children, and everybody with experience must agree that it is a very serious matter to have children infected at the age when they are most open to impressions, at the age which determines the whole future development of their life.

The prevention of Ankylostomiasis is, on the whole, fairly simple, but only if taken up whole-heartedly and thoroughly. The notification of the disease alone is not sufficient, as mostly the cases in the early stages escape diagnosis, and notification in Queensland is only entered into in a most half-hearted way.

It would be necessary, first of all, to map out the infected areas of the disease and where it is found prevalent, make a house-to-house examination, and the sufferers should be compelled, if necessary under penalty, to undergo a thorough treatment.

Then it would be necessary to improve the sanitary arrangements and force the labourers in cane-fields to make use of earth closets, or introduce a portable pan system, taking proper precautions to have the contents properly sterilised.

It is a well-known fact that the eggs of Ankylostoma duodenale require humidity, oxygen, and heat for their development. This gives a clue for the destruction of the larvæ.

Mr. Octavius C. Beale said that not the least interesting part of the discussion was the sincere and strong difference of opinion between the worthy physicians, who had lived and laboured so long in North Queensland, as to the suitability of that country for the Caucasian race.

As Ankylostomiasis was proved to be prevalent amongst white children there, we must remember that recent researches show it to be even more general amongst the young of our race in the Southern States of the American Union. Drinking unboiled water must tend to its spread, and recent experiences in America have shown how very far prophylaxis can go towards the improvement of health and physique under tropical conditions.

The vast volcanic intrusion in the far north, back of Mourilyan Harbour and Cairns, furnishes a glorious tableland, with an invigorating climate and rich soils, whereon Europeans are settling and ought to multiply. At 2,000 feet and 3,000 feet above sea-level the visitor from the South will find healthy women and rosy-cheeked, robust children at all the settlements. It is fortunate for us that such favorable conditions exist over a wide area that will admittedly be occupied by large numbers of our people. The layman cannot observe deterioration—in those districts—whilst we can surely hope that as settlement progresses and farm-products multiply, together with better sanitation amongst an active and intelligent people, there will be continuous improvement.

Offers are being made to Australia of emigrants from Northern Italy, including that Cisalpina which in Roman republican and imperial times supplied a constant stream of bright intellects and citizens of fine physique to sustain the nation. It is still occupied by a vigorous race that might well succeed in subduing from Nature, and defending for the Australian Commonwealth, at least the rich salubrious hill-country of our far North.

INVESTIGATIONS INTO A CASE OF THE LEPROSY-LIKE DISEASE OF RATS.

By J. Burton Cleland, M.D., C.M. (Syd.).
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On the 2nd May, 1910, a rat (*Mus decumanus*) from Ultimo was submitted for examination. The animal proved to be extensively affected with the now well-known leprosy-like disease of rats. There were extensive patches of bare, somewhat thickened, skin on the back, sides, shoulders, and buttocks, with small ulcerated spots. The subcutaneous tissues below these were thickened and infiltrated, and the skin was adherent to them and thus to the muscular tissue beneath. The internal organs showed no changes due to the disease. Smears from the skin lesions and sections of this tissue showed the usual presence of inconceivably vast numbers of acid-fast bacilli. Many cultures were made from the lepra-like areas on to various media, sach as agar, glycerine agar, &c., but in no instance was a growth obtained of acid-fast bacilli, or of a streptothrix up to a period of six months.

Seven rats (*Mus Alexandrinus*), were inoculated with material from the affected rat in the following way :—

Rat 2,492.—The chest was scarified and material from the affected rat rubbed in. A pocket was also made in the same place and a small fragment of leprous material inserted. This animal showed no evidence of disease until its death on the 27th October, nearly six months after inoculation. *Post-mortem examination.*—The base of an incisor tooth was exposed through the lower jaw. No acid-fast bacilli were present in the discharge. There was some scurfy skin on the root of the tail and the adjacent part of the back, but the underlying tissue was not infiltrated. No acid-fast bacilli were seen in this situation. At the site of the pocket in the chest there was a small loose nodule, the size of a grain of wheat, loosely adherent to the skin and subcutaneous tissue. Smears from this area showed numerous acid-fast bacilli, and sections made of the skin over the nodule and of the nodule itself showed a dense mass of acid-fast bacilli in the deeper tissue, with outlying masses around which infiltrated the tissues immediately under the squamous epithelium, isolated bacilli appearing sometimes apparently between the epithelial cells. Sections made of the skin from the root of the tail showed no acid-fast bacilli. Cultures from the lesion were made on glycerine agar and Dorset's egg medium. These nearly all became overgrown in a few weeks. No acid-fast bacilli or streptothrix elements were recognisable up to the end of June, 1911.

Rat 2,493.—Similarly treated to Rat 2,492. This rat died on the 28th November, nearly seven months after inoculation. It was much emaciated. At the site of inoculation was a small encapsuled caseous-looking mass about the size of the tissue originally inserted. Films from this area showed large masses of granular acid-fast bacilli which did not stain well. Sections of the inoculated area showed dense masses of acid-fast bacilli. In the centre of the mass the cells stained poorly and were degenerated. At the periphery there was dense fibrous tissue, showing nuclei taking the stain better. The acid-fast masses seemed more or less confined to the original tissue inserted, except perhaps at the outer edge. Cultures were made from the lesion on sterile rats' flesh with glycerinated broth, on Dorset's egg medium and glycerine agar. At the beginning of July no apparent growth had occurred, but there were still abundant acid-fast bacilli on one glycerine agar tube, and on two of Dorset's egg medium tubes.

Rat 2,494.—Material was inserted into a pocket on the rat's thigh. The animal died on the 11th October, about five months after inoculation. *Post-mortem examination.*—Nothing was noted except a small firmish nodule, the size of a small pea, but elongated, at the site of inoculation. A cloacal fistula was present, the cavity being full of fæces. The distal part of the right horn of the uterus was adherent to the right abdominal wall, and distended in several places with thick curdy pus. Smears from the nodule in the thigh showed vast numbers of acid-fast bacilli, often in dense clumps, only slightly granular; the subcutaneous tissues around the nodule showed many acid-fast bacilli, and a small gland in the groin also showed a few acid-fast bacilli, but as these smears were made after the nodule was incised, they may have come from the latter. Pus from the uterine abscess showed no acid-fast bacilli. Sections of the nodular area were made; the tissue was packed with enormous numbers of the acid-fast bacilli, mostly in dense groups; in one cell a group of non-acid-fast bacilli were seen living. Connective tissue cells were seen in and around the groups of bacilli. There was apparently definite multiplication of the bacilli. Cultures were made on suitable media, but no acid-fast bacilli or streptothrix elements were detected up to the end of June, 1911. Further inoculations into other rats were made from this rat, as detailed later.

Rat 2,495.—A pocket was made in the left leg, and material inserted. The nostrils were swabbed with material, and 3 grams of lepra material given as food, which were eaten by next day. This rat was found dead and highly decomposed on 9th January. *Post-mortem examination.*—The lungs were found occupied by green caseous masses. One or two acid-fast bacilli were found in smears made from the lungs, but these were probably carried in during dissection. At the inoculated site in the left inguinal region a small piece of tissue was found, apparently being that originally placed there and unabsorbed. There was no definite extension to the tissues around. Smears showed vast numbers of acid-fast bacilli, which stained well and were non-granular, and did not look degenerated. Unfortunately, sections were not made of this tissue.

RAT 2,496.—This rat received the same treatment as Rat 2,495. It died on the 25th April. No lesions were detected in the chest, nose, or abdomen. There were enchondromata on the ribs. *Post-mortem examination.*—Smears from the nose showed nothing. The right side of the chest showed numerous degenerated acid-fast bacilli. Sections were made of the skin of the chest, but the site of the lesion was not detected, and thus the origin of the acid-fast bacilli was not found.

RAT 2,497.—A broth emulsion of the acid-fast bacilli from the affected rat was injected into the chest and right and left inguinal regions of this rat. It was also fed similarly to Rats 2,495 and 2,496. On the 12th September, it was found dead and decomposing. Nothing definite was found *post-mortem*. Smears of the left inguinal region and the chest showed numerous acid-fast bacilli which, compared with the original smears, seemed to show less irregular staining. Unfortunately the carcase was thrown away before tissues for sections had been taken.

RAT 2,499.—This rat was injected intra-peritoneally with 2 c.c. of a broth emulsion of the acid-fast bacilli. It was found dead and beginning to decompose on the 31st October. *Post-mortem examination.*—There was no apparent thickening of the peritoneum; no changes were detected in the liver and spleen, in both of which decomposition was evident. The upper lobe of the right lung seemed partly consolidated. Smears from the peritoneal surface showed scattered acid-fast bacilli with a few granules, especially in those made from the spleen and liver. Sections were made of the liver and spleen and of the partly consolidated lung, but no acid-fast bacilli were seen. Sections of a lymph gland, probably the lumbar, were also made. This contained scattered cells full of blackish pigment, probably altered blood. In the centre of one of these masses was a group of acid-fast bacilli having rather a streptothrix appearance. Cultures on Dorset's egg medium and glycerine agar were made from the peritoneum—no acid-fast bacilli were detected up to the middle of November.

RAT 2,500.—This rat was fed with the head, neck, and forelegs of the diseased rat. It died on the 31st March. Nothing was noted *post-mortem*, and smears from the pancreas, abdominal lymph glands and intestine did not show the presence of acid-fast bacilli.

RAT 2,501.—Was fed together with rat 2,500. It was accidentally killed on 31st May, a year after inoculation. The organs all appeared normal, but no smears were taken.

RAT 2,564.—This rat was inoculated in the right groin with small portions of tissue from the small subcutaneous nodule from Rat 2,494 on October 11. It died on October 25, showing nothing of interest in the *post-mortem*. It is to be noted that this rat had been fed on Azoa some weeks before.

RAT 2,565.—This rat was similarly inoculated with a small piece of tissue about the size of half a grain of wheat from the nodule of Rat 2,494, on October 11. It died on 24th March, showing *post-mortem* nothing except collapsed areas in the lung. There were no obvious lesions in the groin. Smear preparations from the lung showed no acid-fast bacilli, but preparations made from the left groin showed fairly numerous, somewhat granular, acid-fast bacilli, and smears from the right groin showed some acid-fast bacilli. Sections of the skin of the groin at the site of inoculation show dense masses of deeply stained and not degenerated-looking acid-fast bacilli in the tissues under the skin in places projecting towards the surface so as to reach the squamous epithelium. These masses are surrounded by living connective tissue cells. In advance of the large groups scattered small groups or an odd bacillus are seen. Below the masses of bacilli is an encapsuled granular non-staining mass surrounded by squamous epithelium whose Malpighian layer is inside. The outer layers of this squamous epithelium have nuclei staining well in places, and poorly—that is, degenerated—in others. Apparently this squamous epithelium has been accidentally implanted during inoculation. In the non-staining central mass are seen one or two acid-fast bacilli. Outside this large mass is another smaller mass of degenerated tissue containing more acid-fast bacilli, and surrounded by large acid-fast masses.

MONKEY, MACAQUE, 2,498, was inoculated as follows :—

(*a*) 3 c.c. of the broth emulsion of the material from the original rat was injected subcutaneously into the back of the waist.

(*b*) A small piece of tissue was inserted into a pocket on the inside of the left thigh; and

(*c*) The left nostril was swabbed with tissue containing the bacilli.

5th July.—The pocket in the thigh has been discharging thin pus containing some acid-fast bacilli since the period of inoculation. The wound has now almost healed, and only a small point is left with a little induration. The monkey has been licking this area. In the groin above the wound is an oval firmish swelling, apparently with a softer centre, about the size of a walnut, with some induration around it, which extends as a cord outwards below Poupart's ligament.

11th July.—There is a dark pigmentation and a little thickening at the seat of the pocket in the thigh. The groin above has been discharging thick curdy white pus, and has now much decreased in size, but still shows definite thickening. Smears of pus show besides other bacilli, occasional small groups of acid-fast bacilli, apparently thicker and more regularly stained than these bacilli usually are. Over the seat of inoculation in the back is a bare area the size of half-a-crown, with a fluctuating swelling below it.

21st July.—The groin is still discharging a little, and there is still definite thickening along Poupart's ligament. The pus did not show any acid-fast bacilli, but a few rounded deeply-stained acid-fast spore-like bodies. The area in the back is now the size of a walnut, and discharging curdy slimy pus from several orifices. Smears from this pus show few degenerated scattered acid-fast bacilli and occasional rounded acid-fast spore-like bodies as well as a few gram negative bacilli.

16th August.—There is only a little thickening now remaining at the seat of inoculation in the thigh and in the groin. The area in the back is still swollen and moderately firm with very little discharge, but probably a deep pocket of pus.

5th September.—The areas in the thigh are apparently normal and with very little if any thickening. The area in the back still shows a little curdy pus which is very stringy and exudes on pressure. Smears show very occasional faint granular acid-fast bacilli and some cocci.

By December the lesions in the groin and the back were practically quite healed.

9th March, 1911.—No lesions could be detected anywhere.

This same monkey was also inoculated on 11th October with material from the small nodule from Rat 2,494, as follows :—

(*a*) A small piece of tissue about the size of half a grain of wheat was placed in a pocket in the right groin, and

(*b*) An emulsion of the tissue was injected into the right groin subcutaneously.

12th November.—There was a large abscess at the site of inoculation in the groin.

28th December.—This abscess was much reduced, though with still a little pus and a thickened scar, whilst the site of injection of the emulsion in the right loin showed nothing.

9th March.—No lesions were detected.

The monkey died on the 10th July. There was no definite change as regards thickening, &c., at any of the sites of inoculation, and films from these areas did not reveal the presence of any acid-fast bacilli.

Smears made from the left nostril, which had been swabbed with an emulsion, showed a number of acid-fast bacilli usually in pairs, end to end, and much larger than the leprosy-like bacilli used for inoculation. Sections of the mucous membrane of this nostril did not reveal the presence of any of these acid-fast bacilli in the tissues. Cultures on Dorset's egg medium and on glycerine agar have failed to show a growth so far of these acid-fast bacilli. None of these bacilli were seen in a swabbing from the right nostril.

Four guinea-pigs were inoculated with the original lepra material, but these had all died within the period of one month without showing any lesions of a lepra-like nature.

Four rabbits were also inoculated on the 2nd May, as follows :—

RABBIT 2,488.—Belly scarified and an emulsion rubbed in and nostrils swabbed also with an emulsion. This animal died on 21st October; there was much emaciation. There was a bare area where the hair had been plucked out of the abdomen and the lepra material rubbed in. This was surrounded by an area from which the hair had also been plucked out or cut, in which there was a short length of hair. Nothing was noted in the nostrils. No acid-fast bacilli were seen in smears made from the bare batch on the abdomen or from the nostrils, and sections of the skin-area were likewise negative.

RABBIT 2,489.—Was similarly treated, and died on the 10th February. No lesions were detected anywhere.

RABBIT 2,490.—Received a piece of tissue in a subcutaneous pocket and its nostrils were also swabbed. It died on 4th July, and showed no disease of the viscera, but a dryish ulcerated area the size of sixpence at the site of inoculation in the thigh. Smears from this area showed some acid-fast bacilli, but these were not seen in the nostril smears. Sections of the ulcer did not show any invasion by acid-fast bacilli.

RABBIT 2,491.—Was similarly treated and died on 31st October. At the post-mortem, nothing pathological was detected beyond some large cysticerci. Smears from the inoculated areas did not reveal the presence of acid-fast bacilli.

Dr. Tidswell (Report of the Board of Health on Leprosy in New South Wales, 1904, p. 13) has already recorded the previous occurrence of a case of this leprosy-like disease in a rat, *Mus decumanus*, from this State. It is interesting to note that in both these instances the rats were of this species.

During the last ten years probably nearly 300,000 rodents* have been systematically examined in Sydney, and it would therefore appear that the occurrence of this disease in such a marked degree as to be easily recognisable is very rare. It is interesting to note that this leprosy-like disease of rats would appear to be nearly as rare in the rats of this State as is the true leprosy amongst the human population of the State. I do not wish to discuss at present the question as to the exact connection between the disease of rats and the disease of man. It seems to me, however, obvious that in both these diseases the means by which the infection of the patient is secured are factors of very rare occurrence, either because the chance of infection is very small or else that the means by which infection is obtained requires a chain of circumstances which is very rarely secured. It may be, of course, that in both instances, as in the case of actinomycosis, the organism normally has an external saprophytic existence, and that only occasionally does it have an opportunity of exerting its pathogenic effect upon animals or man. This perhaps would explain the very rare sporadic appearance of each disease. In rats, as in man, it would seem that the disease is one of slow progress, and as the average life of the rat is so much smaller than the average life of man, the likelihood of the disease developing into a stage easily recognisable in rats is less great than in the case of man. It may, therefore, be that more rats than we believe are infected and that the lesions produced by the presence of the bacilli often have not extended sufficiently greatly to be recognisable with ease.

I am inclined to think that in the disease in rats the lesions are chiefly due to the enormous multiplication of the bacilli with consequent interference with the nutrition of the parts involved, and hence the slow development of the lesions. If this be the case it should be possible to demonstrate occasionally the presence of acid-fast bacilli in similar situations in rats, which did not as yet show marked signs of the disease. With this object in view, several hundred smears from the subcutaneous tissues of the rats, systematically examined by us, have been searched for acid-fast bacilli. In only one instance were a few acid-fast bacilli seen; and in this case, unfortunately, the use of an imperfectly cleaned slide could not be excluded, especially as the few bacilli seen were not unlike those obtained from a tuberculous sputum of a human being.

I am much indebted to the courtesy of the Director of the Bureau of Microbiology for permission to publish the details of this case.

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* From April 18, 1900, to March 31, 1903, 46,775 rodents were examined. From May 1, 1903, to the end of that year, 17,180 rats and 10,880 mice, and from the last date till the end of 1910, 61,530 *Mus decumanus*, 63,891 *Mus rattus* and *Alexandrinus*, and 87,771 *Mus musculus* were examined. Total rodents 288,047, of which probably less than one-third were *Mus decumanus*.

INSECTS AND THEIR RELATIONSHIP TO DISEASE IN MAN IN AUSTRALIA.

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In discussing the question of insects in their relationship to disease in man, it has seemed to me of value to deal with the subject systematically, taking the various classes of insects in turn. Further, the term "disease" has been taken in its broadest meaning,—that of disease, a departure from normal well-being—thus including any reactive process in man due to mechanical, chemical, or other stimuli originating from insects. From this wide standpoint, I have collected together such references as I have been able to obtain of insects in relation to disease in man in Australia, and have added to these such observations as I have been able to make myself.

NEUROPTERA.

Some of our Australian termites can grip hold of human skin with their jaws, and where the skin is delicate, can just make themselves felt. This bite is so feeble, however, that from the medical aspect it can be neglected.

Family Psocidæ (*Book-lice*).—The *Age* in Melbourne recently published a short note saying that vast armies of small soft-bodied insects belonging to the genus *Psocus* were descending on that city. The writer says they give a sharp nip without after-effects, and were first noticed in the State near Canterbury, two years ago. Now they had reached Camberwell. Although only the size of a pin-head, their presence in a house is easily detected as they brush from books on to human beings, where they make themselves felt. Mr. C. French, junior Assistant Government Entomologist, had received complaints from many parts of the State concerning them.

HYMENOPTERA.

This order includes the various bees and wasps and the ants.

Formicidæ (*Ants*).—The number of species of ants in Australia is very considerable, and amongst them are some formidable species. There seem, however, very few records of annoyance or injury to man as the consequence of the bites of these insects.

Green Tree Ants (*Oecophyllus smaragdina*).—In the tropical parts of Australia these insects seem to be exceedingly numerous in places. Presumably it is this insect that is referred to by Flinders when he was in the Gulf of Carpentaria, in February, 1803. Flinders says that "Most of the bushes contained nests made by the small green ant; and if the bush was disturbed these revengeful little animals came out in squadrons and never ceased to pursue until the disturber was out of sight. In forcing our way out of the underwood, we sometimes got our hair and clothes filled with them, and as their bite is very sharp, and their vengeance never satisfied, there was no other resource but to strip as expeditiously as possible."

Mr. S. W. Jackson, in 1909 (*The Emu*, Vol. 8, Part 5, June 1909, p. 245), thus refers to the insect in question: "The green tree-ant (*Oecophyllus smaragdina*) I found plentiful on these islands (Dunk Island, off the Queensland coast), and on the coast of North Queensland generally. While up at a nest of the Nutmeg-Pigeon in a mangrove tree I got covered all over with these green insects, and received many bites from them. Their nest, which is about the size of a football, and placed in a tree, is composed of green leaves, which are all neatly fastened together with a white substance resembling cobweb."

Of the other Hymenoptera the wasps and bees deserve notice, though few references to the effects of the stings of Australian species exist in literature.

Dr. George Bennett, in "Wanderings in New South Wales" (1834), Vol. 1, p. 328, refers to a young black who was stung by a wasp, and says: 'He no doubt suffered severe pain; he threw himself upon the ground and rolled about, but no sound escaped his lips.

Sir T. L. Mitchell, in "Three Expeditions into the Interior of East Australia," 1838, Vol. 1, p. 104, describes being attacked, near the Gwydir River, by a species of wasp which he described and named *Abispa australasiana*. The insects, which attacked another man and him self, caused a sudden extreme pain in the thigh in his case. The effect of the sting was so permanent that when he alighted in the evening on that leg from his horse, not thinking of the circumstances, he fell to the ground "the muscles having been generally affected. The wound was marked by a blue circular spot, of the size of a sixpence, for several months."

I have also been informed of an instance of death, apparently from œdema of the larynx, through a man drinking out of the spout of a water-bag in which a wasp was at the time drinking and hidden from view. It must have been taken into the mouth and stung him, and he apparently died from suffocation.

The only bees which seem of importance from their bite are the common introduced honey bees.

LEPIDOPTERA.

As is well-known in various parts of the world, certain caterpillars have hairs which possess a very irritating effect on the human skin Mr. J. O. Tepper, F.L.S., of the Adelaide Museum, informs me that in the case of species of *Teara* and *Nola*, the hairs, though flexible when moist, become very brittle when dry, breaking into small fragments which easily fly about, and gaining access to the neck and wrists, cause intense irritation. Handling the "nest" or even opening a bottle containing portions of this—cast skins and excreta—may produce these results, the fragments being so fine. A very interesting article entitled "Bag-shelter Caterpillars of the Family *Liparidæ* that are reputed to kill Stock," is contributed by Mr. W. W. Froggatt, F.L.S., to the *Agricultural Gazette of New South Wales*, May, 1911. It would appear from information gained from these two sources, that these processional caterpillars belong to the family *L. paridæ;* to this family also belongs the notorious Gypsy Moth (*Portiretria dispar*) of North America, so destructive to trees there, which also possesses irritating hairs; and that they form bag-shelters in the trees, which they inhabit by day and feed at night, leaving their cast skins (with the hairs) and excreta in the nest. Mr. Froggatt says that the species *Teara contraria* is common on the weeping myall (*Acacia pendula*) in western New South Wales. "The hairs are very irritating, and two men engaged near Scone in cutting down the nests were so badly attacked by a painful and serious rash that they had to go to hospital. Another species in New South Wales is *Ocinaria Lewini*. It has done much damage along our South coast, and near Armidale. The grubs, getting among the grass, were eaten by stock, especially horses, causing swelling and ulceration of the lips, tongue, &c., causing death in many cases from starvation. On human beings they cause great itching and tenderness, and one man was reported to have lost an eye. A child, as a result of secondary infection, required lancing of its hand three times."

Mr. Tepper informs me that another processional caterpillar, *Nola lugens*, makes similar nests and has the same irritating properties. Black-fellows will not climb trees when the bag-shelters of the processional moths are in them.

Mr. Tepper says also that the caterpillars of the genus *Doratifera* (*D. vulnerans*, *D. oxleyi*, and *D. longerans*), when irritated throw out six clusters of yellowish spines, which, coming in contact with delicate skins, cause considerable irritation.

DIPTERA.

The various two-winged insects, flies, mosquitoes, sand-flies, &c., may be viewed from three quite separate aspects as affecting the health of man. These are shortly :—

1. The growth of larvæ in human tissues.
2. Bites of insects. This includes not only the merely mechanical effects of the bites, but also the question of the transmission of parasites, especially protozoa.
3. The contamination of food-stuffs, &c., by means of flies, with which may be included direct infection of wounds, &c., and of the conjunctiva by organisms carried by the flies, as well as the mere mechanical annoyance of these insects when in great numbers.

1. The Growth of Larvæ in Human Tissues.

The deposition of the eggs or living maggots of flies in wounds and open sores very frequently occurs in animals in Australia. In the case of sheep economic loss from this aspect is well known to be extensive. In these animals the maggots are not only deposited in wounds, &c., but also in the soiled wool around the anus. Mr. Froggatt's investigations indicate that the flies responsible are not so much a species of *Lucilia*, such as *L. cæsar*, but *Calliphora oceani* and *C. villosa*, these being apparently identical with *Anastellorhina augur* and *Pollenia stygia*, identified for me by Mr. Austen.

I have also seen a wound in a hawk infested with maggots, and Colonel Warburton in his "Journal of Exploration," p. 240, scooped maggots out of fly-blown sores on a camel's back with a pint-pot in Western Australia. There are unquestionably numerous other instances in which animals in Australia have been affected in this way.

The references to the occurrence of living maggots in human beings in Australia are few. The earliest appears to be a reference by Dr. George Bennett in "Wanderings in New South Wales," (Vol. 1, p. 294), who refers to a case of myiasis of the ear. He says: "During my journey a man on one of the stations complained to me of a dull pain in his ear, and as if something were moving in it; . . . by pouring brine into the ear a large white maggot crept out, and afterwards some smaller ones."

In the *Australian Medical Journal* for 1865 (Vol. 10, p. 95), mention is made of twenty maggots being removed from the ear of a little boy at Castlemaine, Victoria.

In the *Australasian Medical Gazette* for March 1895, p. 97, Dr. G. H. Salter, of Ballan, Victoria, described four cases of myiasis in human beings, in one of which the eye was affected; in another the nose of a child; in the third a child's navel; and in the fourth a malignant growth of the temporal region. The cases are as follow:—

"An alcoholic woman came complaining of pain in the eye, and a feeling as though little pebbles were rolling over the eyeball. The eye was red and much swollen, the lids more or less glued together with pus and blood. On separating them the space between the lower eyelid and the globe was teeming with maggots, about twenty to thirty in number, apparently, from their size, two or three days' old. The cornea was cloudy and ulcerated at two or three spots, and vision was materially impaired two months later."

In Gisborne, a few years previously, Dr. Salter removed a number of full-sized maggots from the nose of a child of seven months, the subject of hereditary syphilis. On another occasion the presence of two maggots accounted for a discharge from a child's navel.

In a woman with epithelioma of the left temporal region, a large number of full-grown maggots were removed which had almost demolished the growth as well as the left lower eyelid, and had found their way into the left orbit.

In the same publication (May, 1895, p. 213), Dr. Russell, of Adelaide, described a case in which twenty-three maggots were removed from between the eyelids, and a case in which five maggots were extracted from a boy's ear, from which there was a fœtid discharge. In describing the case of maggots from the eyelids, he says: "There was a rounded open ulcer the size of a sixpenny piece at the inner corner of the eye, which had not ulcerated through the whole thickness of the lid. There was much conjunctivitis, but no ulceration."

The Bureau of Microbiology also possesses an old museum specimen consisting of maggots removed from an ulcer in a leper at the Coast Hospital. These Mr. W. W. Froggatt has identified as those of a blow-fly. I have also, about fourteen years ago, at the Royal Prince Alfred Hospital, seen a patient with a deep syphilitic ulcer of the leg, in the depths of which were numerous large maggots.

2. The Bites of Insects (Diptera).

Six groups of biting flies, and the sub-order Siphonaptera, come under this heading, viz.:—

(*a*) *Simulidæ*
(*b*) *Chironomidæ* } both of which are called sand-flies.
(*c*) *Culicidæ*, which consists of the mosquitoes, the transmitters of filaria, malaria, and probably dengue.
(*d*) Biting flies of the family *Leptidæ*.
(*e*) The large biting flies belonging to the *Tabanidæ*.
(*f*) Biting flies of the family *Muscidæ*.
(*g*) The biting flies of the family *Hipposboscidæ*.
(*h*) Fleas (*Siphonaptera*).

(*a*) and (*b*), *Simulidæ* and *Chironomidæ* (Sand-flies).—The only work on the Australian species of those families of Diptera, popularly known as sand-flies, is that by F. A. A. Skuse, in the "Proceedings of the Linnean Society, New South Wales," Vols. 3 and 4, Second series, for 1888 and 1889.

He places the Australian Sand-flies in two families, viz., the *Simulidæ* and the *Chironomidæ*. Of the *Simulidæ* he was only able to obtain one species, which he has named *Simulium furiosum*. In reference to this species, he says: "I have only twice found specimens of the following described species, and then only in very limited numbers, and I know of no other instance of its capture, which seems rare and local. The so-called sand-fly of this country is the species of *Ceratopogon*, or probably more than one species, only too abundant in many places, and inflicting many painful bites." His specimens were taken at Gosford and Berowra, in New South Wales, in August and September.

Of the genus *Ceratopogon*, belonging to the Chironomidæ, he says: "The families of several species of Ceratopogon are blood-suckers, and capable of inflicting very painful wounds. These annoying insects are particularly numerous in Australia, and generally go by the name of sand-flies. Their bites are quite as severe as those of the mosquitoes, and I am told that in some parts of this country the cattle are dreadfully attacked by them, and are sometimes driven frantic by the irritation of their wounds." The larvæ of Ceratopogon reside in water, in the ground, in manure, in the bark of decaying timber, &c. Some are said to be carnivorous, devouring the larvæ and pupæ of other insects.

Of the genus Ceratopogon, Skuse described seventeen new species and refers to another Australian species (*C. rhynchops*, described by Schiner). The only species which he specifically states as biting man is *Ceratopogon* (now *Culicoides*) *molestus*. His remarks as regards this species are as follows: *Habitat:* Sydney, and generally in New South Wales (Masters and Skuse), Como, N.S.W. (Dr. Katz), common from December to April. *Observations:* Generally called sand-flies, and a particularly annoying insect in many localities. Some specimens in spirit received from Dr. T. Bancroft, from Brisbane, seem to belong to this species, which is partly allied to the European *C. arcuatus*, Winn.

C. molestus is common in the coastal districts of New South Wales, its bite being very irritating and leaving a small raised lump, itching for several days in some cases.

LIST OF MOSQUITOES RECORDED IN AUSTRALIA.

The following is a list of mosquitoes and localities so far recorded, as far as I can ascertain, for Australia. It is compiled from Theobald's, Giles's, Blanchard's, and T. L. Bancroft's works or papers. Those marked with an asterisk are fully described by Dr. T. L. Bancroft in his "Monograph on the Mosquitoes of Queensland," published in the Annals of the Queensland Museum.

**Anopheles corethroides*, Theobald	Burpengary and Alderley, Queensland.
Anopheles stigmaticus, Skuse	Blue Mountains.
**Pyretophorus atratipes*, Skuse	South Queensland, from Caboolture to Moreton Bay and Enoggera; Berowra, New South Wales.
**Myzorhynchus bancrofti*, Giles	Caboolture to Enoggera, South Queensland.
**Nyssorhynchus mastersi*, Skuse (= *Anopheles musivus*, Skuse; Theobald gives *muscivus*).	South Queensland, Sydney.
**Nyssorhynchus annulipes*, Walker. (Bancroft considers *N. mastersi* to be the starved form.)	Tasmania, South Queensland, New South Wales, Port Darwin.
**Toxorhynchites speciosa* (*Megarhinus speciosus*), Skuse.	Port Denison, Mackay, Queensland; Port Darwin, and Kuranda, Thursday Island, North Percy Island, Caboolture to Moreton Bay and Enoggera; doubtful species from Sydney (Skuse).
**Mucidus alternans*, Westwood (= *Culex alternans*, Westwood, *C. commoveus*, Walker, and *C. hispidosus*, Skuse.)	"Hexham grey." Common all over Queensland, especially along the coast; New South Wales, Natal (Walker), New Guinea, Johnstone River, North Queensland, Port Darwin.
**Stegomyia fasciata*, Fabr. (= *C. bancrofti*, Skuse, var. *Queenslandensis* mentioned by Giles. Blanchard says the species varies even in the same locality.)	New South Wales, Queensland, Victoria (*vide* Giles).
**Stegomyia punctolateralis*, Theobald.	Brisbane, rather rare; Phillip Island.
**Pseudoskusea multiplex*, Theobald	Queensland, New Guinea, wide range in South Queensland.
**Scutomyia notoscripta*, Skuse. (*Culex notoscriptus*.)	Sydney, Queensland, New Guinea.
**Macleaya tremula*, Theobald	Deception Bay, Queensland.
**Gilesia aculeata*, Theobald	South Queensland.
**Grabhamia flavifrons*, Skuse	Brisbane, Blue Mountains.
**Culicada bupengaryensis*, Theobald.	Burpengary, South Queensland.
Leucomyia australasiensis, Theobald	Stannary Hills, Queensland.

*_Culicelsa annulirostris_, Skuse	Blue Mountains, Queensland.
*_Culicelsa vigilax_, Skuse. (= _C. marinus_, Theobald).	New South Wales, Queensland, Johnstone River, North Queensland.
*_Culicelsa alboannulatus_, Macquart	East Coast of Australia, New South Wales, and Queensland.
*_Culex fatigans_, Wiedemann (Sub-species _macleayi_, Skuse, mentioned by Giles, Blanchard.) (Sub-species _skusii_, Giles, given by him as _C. sp. prope, C. ciliaris_, Skuse. [Skuse describes it as _Culex_ (? sp.), and at the end says "possibly a variety of _C. ciliaris_, Linn., i.e., _C. pipiens_, Linn.])	Queensland, New South Wales.
*_Culex linealis_, Skuse (Theobald gives _linearis_.)	New South Wales.
*_Culex_ (?) _tigripes_, Grandprè et Charmoy. (_Culex maculicrura_, Theobald.)	Queensland and foreign.
Culex atripes, Skuse	Homebush, Sutherland, Knapsack Gully.
*_Culex vittiger_, Skuse (Theobald gives _vittager_.)	New South Wales, Queensland, Pt. Denison to Gosford on seaboard.
*_Culex procax_, Skuse	New South Wales, Queensland, Gosford, South Clifton, Burpengary.
*_Culex rubithorax_, Macquart	Tasmania, South Queensland, Phillip Island; round Moreton Bay, not plentiful.
*_Culex occidentalis_, Skuse	Somerville and Healesville, Victoria; King George's Sound, South Queensland, Mt. Victoria.
Culex camptorhynchus, Thompson (Giles says Skuse suggests synonymous with _Culicelsa alboannulatus_.)	Sydney.
Culex frenchii, Theobald	Victoria.
Culex sagax, Skuse	Murrumbidgee, Burpengary.
Culex australis, Erichson (= _C. crucians_, Walker.)	Tasmania, Maryville, Victoria.
*_Culex cylindricus_, Theobald	South Queensland.
*_Culex pseudomelanoconia_, Theobald	South Queensland.
Culex nigrithorax, Macquart	Tasmania.
Culex oceaneus, Theobald (Mentioned by Giles.)	Queensland.

**Chrysoconops acer*, Walker ...	New Zealand, Queensland.
**Mansonia uniformis*, Theobald ...	Africa, India, Madagascar, Phillip Island, New Guinea, Australia, Port Darwin.
**Finlaya poicilia*, Theobald... ...	New Guinea, Penang, Phillip Island.
Finlaya kochi, Dünitz	New Guinea.
Ædes venustipes, Skuse. (*Ædeomyia*(?) *venustipes*.) (Theobald says probably belongs to *Finlaya*.)	Elizabeth Bay, Sydney.
* *Skusea funerea*, Theobald ...	Deception Bay.
**Uranotænia pygmæa*, Theobald ...	Deception Bay, Burpengary, Enoggera.
**Anisocheleomyia nivipes*, Theobald.	Queensland.

PERSONAL OBSERVATIONS ON MOSQUITOES.

The following species of mosquitoes, collected in New South Wales and South Australia, were forwarded to Mr. E. E. Austen, at the British Museum, Natural History Branch, and through him were identified by Mr. Edwards, who is in charge of the collection of these insects. To these gentlemen I am greatly indebted for the identification or confirmation of the species.

1. *Nyssorhynchus annulipes*, Walk.

This mosquito, though not a common one in the neighbourhood of Sydney, is not on the other hand rare. So far, I have not found it biting. The imagines have been found just at sunset trying to escape out of the closed windows of houses at Neutral Bay, and at the Hawkesbury River, apparently having been hidden away in the houses during the day. The adults have been bred from larvæ obtained at Neutral Bay, at Richmond (November), and at Locksley, near Bathurst (February). Adults have been found at the Hawkesbury River, in November.

2. *Mucidus alternans*, Westw.

This very handsome and striking species seems widely distributed along the coastal districts. It can be a severe but leisurely biter. Specimens have been caught at Hawkesbury River Bridge (January), Berry (January), and Botany Bay (March). It bites by day.

3. *Scutomyia notoscripta*.

This handsome little insect is a severe biter and a day flyer. It may be found in the scrub or near habitations. I have only found it once inside a house caught on a sheet of tanglefoot. Specimens have been collected at Middle Harbour, Sydney (November), at Neutral Bay (December), and at Manly, Sydney (February). I have also obtained it at Adelaide.

4. *Desvoidya* sp.

This was obtained at the Hawkesbury River Bridge in March. This seems to be the first record of this genus from Australia. *D. obturbans*, Walker, has been recorded from New Guinea.

5. *Pseudoskusea multiplex*, Theob.

A specimen of this mosquito was obtained, biting, at Kurnel, Botany Bay, in March.

6. *Culicada westralis*, Strickland (MSS).

Specimens have been bred from salt water at Port Adelaide in May. This is apparently the mosquito so abundant in the ti-tree scrub over salt-water marshes in this district, biting by day. Specimens of this species were also obtained by me in May, at Tailem Bend, near Murray Bridge, in South Australia, biting severely at sundown. Mr. Edwards, in identifying my mosquitoes, says of these specimens: "They appear to belong to this species, which according to Mr. Theobald, will shortly be described in the "Entomologist."

7. *Culex annulirostris.*

I have only obtained one adult of this species, secured at Manly, Sydney, in February.

8. *Culicelsa vigilax*, Skuse.

This is essentially a scrub mosquito, biting by day and also after dusk. It may be present in myriads in certain parts, especially after rains. It has been obtained at the Hawkesbury River Bridge from October to March, and at Middle Harbour, Sydney, in February.

9. *Culicelsa albo-annulatus*, Macq.

So far I have not captured this species in the adult stage, but have bred it from larvæ obtained at Rydalmere, Sydney, in November; at Tent Hill, New South Wales, in December; at Neutral Bay, Sydney, in January; and at Locksley, near Bathurst, in February.

10. *Culex fatigans.*

This species is very common at certain seasons of the year, being much more numerous after abundant rain. It seems exclusively a night biter, remaining at rest in dark cupboards, &c., during the daytime. At night-time its biting is not severe but very irritating, leaving on many people minute reddish papules. On myself its bite leaves no mark whatever, and the irritation from it soon ceases. It has been bred from larvæ, at Richmond, in November; has been found at Neutral Bay, from December to March, being very abundant indeed after rain. I have obtained it at the Hawkesbury River Bridge, in March. A few specimens of a paler form were obtained in Adelaide, in May.

11. *Culex procax.*

Specimens which appear to be this species have been obtained at Hawkesbury River Bridge, biting in October, November, and March. Other specimens of this species have been obtained at Berry, biting by day, in June.

12. *Culex rubi-thorax*, Macq.

One specimen of this species was obtained at Neutral Bay, Sydney, n January.

13. *Culex occidentalis*, Skuse (?)

A male and male were bred at Milson Island in November

14. *Culex australis*, Erichs.

At the beginning of December some specimens of this species were obtained at the Hotel Kosciusko, at a height of 5,000 feet, in a wood, biting during the daytime. The larvæ were found in a shallow pool of water at a height of 6,000 feet. Close besides this pool of water was a snowdrift. The shallowness of the water in the pool, however, allowed it to become almost lukewarm as a result of the sun's heat, which gave the larvæ an opportunity to develop. Imagines were bred from these larvæ.

(*d*) *Biting Flies of the Family Leptidæ.*—In June of this year (1911), on the upper parts of an unfrequented arm of Middle Harbour, near Sydney, several flies, with a somewhat mosquito-like action when flying, settled upon my hands and began biting. Several of these I secured whilst beginning thus to feed, and forwarded specimens to Mr. E. E. Austen, of the British Museum, who finds they belong to the family *Leptidæ*, and to a genus near *Symphoromyia* and apparently represent two distinct species, both undescribed. He adds, further: "The species belonging to the family *Leptidæ* prey upon other insects, but do not as a rule suck vertebrate blood. The blood-sucking habit, however, is said to have been observed in this family in a few instances, including a Californian Symphoromyia, and the common European *Leptis scolopacea*, Linn., both sexes of which are said to have been observed to bite in France, although neither sex has ever been known to do so in the British Islands."

Since I captured my own specimens, Dr. Cox, of Helensburgh, has sent me a similar biting fly from that neighbourhood, and Mr. Gallard, of the Agricultural Department, has met with them near Gosford (Ourimbah), in June. The latter says: "They frequent the scrubby land where the wallabies are generally found, and we call them wallaby flies. If you bare any part of you, they attack you like a mosquito, and their bite is far worse."

(*e*) *Tabanidæ.*—A number of species of these biting flies have been described from Australia, but so far none of the species have been identified with the spread of disease in man or in animals. The introduction of trypanosome infections of animals might, however, from the presence in parts of considerable numbers of these flies, be fraught with danger.

Mr. Froggatt informs me that he has a list of the Australian Tabanidæ so far recorded now in process of publication by the Government of New South Wales. I myself have met with the following species, which have been kindly identified for me by Mr. E. E. Austen, of the British Museum.

Pangonia tricolor, Walker. This was collected in 1906, at Kelmscott, in West Australia, where it is a common species, and is a severe biter.

Tabanus, near *T. antecedens*, Walker. This was stated by Mr. Austen to be not hitherto represented in the British Museum collection. It came from the Darling Ranges, West Australia, 1906.

Tabanus vetustus, Walker. This was taken at Encounter Bay, South Australia, where it occurs along the sea-shore, and bites man severely.

Tabanus abstersus, Walker. This I have taken near Sydney.

Tabanus sp. incert. has also been taken near Sydney.

It may be interesting just to mention the names of the following species referred to by Mr. W. W. Froggatt in his "Australian Insects."

Pangonia guttata (1 inch in length), coastal forests; *P. rufovittata*, wide range over Australia, especially near Sydney; *P. auriflus*; *P. concolor*, Walk.; *P. violacea*; *Tabanus brevidentatus*, Macq.; *T. edentulus*.

Mr. Froggatt in this work refers to an outbreak of malignant pustule in cattle in New Caledonia, said to be traced to an undetermined species of *Pangonia*.[1]

At the September meeting of the Linnean Society of New South Wales, Mr. Froggatt exhibited also the following additional Australian species of tabanids:—*T. nigropictus*, Meig.; *T. ardeus*, Meig.; *T. regisgeorgeii*; *T. Jacksonii*, Macq.; and *T. gregarius*, Erichs.

Eyre in his "Explorations" (Vol. 1, pp. 318, 338), mentions meeting with great numbers of a tabanid—"a very large greyish kind of horse-fly, with a huge proboscis for sucking up the blood"—along the Great Australian Bight in 1841. From 12th to 18th March, he says, "the flies were very numerous, troublesome, and irritating tormentors. They literally assailed us by hundreds at a time, biting through one's clothes, and causing us constant employment in endeavouring to keep them off. I have counted twenty-three of these bloodsuckers at one time upon a patch of my trousers 8 inches square."

(*f*) *Biting Flies of the Family Muscidæ.*

There are, of course, no tsetse flies in Australia, and the only biting member of this family that I know of is *Stomoxys calcitrans*.

Stomoxys calcitrans, Linn.—This is a well-known biting fly, sometimes called the stable fly, of more or less world wide distribution. It can be at once distinguished from the domestic fly by the long biting proboscis, and by the sunken looking whitish patch on the thorax. It is common around Sydney, Melbourne, Adelaide, and Perth, and I have met with it in nearly all the country districts in New South Wales where I have been.

The flies can frequently be noticed sunning themselves on fences, &c., when gorged with blood, in the neighbourhood of dairies, stables, and houses. It is often found in the centres of cities, and in some of the suburbs large numbers are present as in parts of Sydney. It occasionally bites man, but appears to prefer feeding on horses and cattle. It is the cause of their continually whisking their tails, showing restlessness, and moving about. It is a species which might easily transmit anthrax from an animal, in the septicæmic form of this disease, to others.

The following reference in the *Australian Medical Journal* (Vol. 10, 1865, p. 174), under the heading, "Poisonous House-flies," probably refers to *Stomoxys calcitrans*, if a bite were really inflicted. It would seem to be a case of infection, perhaps of malignant œdema, from organisms conveyed by the fly. The account is as follows: "The injurious consequences resulting from the irritating punctures of these domestic pests are thus referred to in a recent number of the *Newcastle* (N.S.W.) *Chronicle:* "James McConnell, baker and drayman, of Bolton-street, on Friday, 20th instant (May ?), was stung by a house-fly on a small sore, on the knuckle of the middle finger of the right hand. The hand immediately began to swell, and, although medical assistance was at once obtained and remedial measures applied, by the next morning the whole of the hand and arm were swollen to an enormous size, and the inflammation was extending itself to the trunk. Dr. Bowker, by the free application of caustic to the hand and arm succeeded in arresting the progress of the inflammation, which has now almost subsided, although the back of the hand is still considerably swollen."

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[1] Megnin and Germain, Bull. Soc. Govt. France, vol. viii. sec. 5.

(g) *Bites from Flies of the Family Hippoboscidæ.*

Several of the early explorers refer to meeting with a fly which they call the "Kangaroo-fly." From Sturt's description of it, Mr. Froggatt tells me he is inclined to think that the fly, in this case at least, was *Ortholfersia Macleayi*, Leach., one of the Hippoboscidæ. Mr. Froggatt says that these flies will frequently leave a wallaby that has been killed and fasten themselves to the dog's nose, causing it much annoyance. Sturt's description ("Two Expeditions into the Interior of Southern Australia," Vol. 1, p. 71), when camped in January, 1829, at New Year Range, between the Macquarie Marshes and the Darling, is as follows: "Our camp was infested by the kangaroo-fly, which settled upon us in thousands. They appeared to rise from the ground, and as fast as they were swept off were succeeded by fresh numbers . . . . The men were obliged to put handkerchiefs over their faces, and stockings upon their hands; but they bit through everything . . . To add to our discomfort, the animals were driven almost to madness, and galloped to and fro in so furious a manner that I was apprehensive some of them would have been lost. I never experienced such a day of torment; and only when the sun set, did these little creatures cease from their attacks . . . The kangaroo-flies (*Cabarus*) . . . were about half the size of a common house fly, had flat brown bodies, and their bite, although sharp and piercing, left no irritation after it." The insertion of the word "*Cabarus*" after kangaroo-flies is perhaps a misprint for "*Tabanus*"—the description of the fly is, however, not like that of one of the Tabanidæ.

Eyre ("Explorations," Vol. II, p. 56), near Cape Arid, on the southern coast of Australia, under the date, 26th May, 1841, refers thus to a kangaroo-fly—perhaps the same species: "In the evening, the kangaroo-fly (a small brown fly) became very troublesome, annoying us in great numbers, and warning us that rain was about to fall." This fly may, however, be one of the Leptidæ previously referred to.

Of other members of this family, it way be mentioned that, in 1907, when camped on the Strelly River, in North-west Australia, examining some camels recently imported, Mr. H. M. Giles, my companion, identified *Hippobosca camelina* and *H. equina*, which were caught near these animals and frequently annoyed the horses. I am not aware that they ever attached themselves to man. The same may be said of the common sheep tick (*Melophagus ovinus*).

(*h*) *Fleas* (*Siphonaptera*).

The study of fleas has naturally increased greatly in importance since the discovery of the rôle they play in the spread of plague. It is unnecessary to dwell upon this point here, save to state the species which have been found on rats in Australia. These comprise the following :—

Lœmopysylla cheopis.—This is common at certain seasons on rats, and is known to bite man, thereby transmitting plague in certain given circumstances. It has been found biting man in Sydney.

Ceratophyllus fasciatus.—This is occasionally found on Australian rats. I have come upon the species in West Australia, and it is fairly common in Sydney.

Ctenopsylla musculi.—This a common flea on rats, and apparently rarely, if ever, bites man.

Ctenocephalus canis.—I have found this species very abundant indeed in the suburbs of Perth, such as Cottesloe, towards the end of the year, biting frequently. Children are sometimes covered with the bites, and in consequence suffer much irritation. This species was kindly identified for me by the Hon. N. Charles Rothschild. The same species is common in Sydney.

Pulex irritans.—This species is well known in Sydney.

BUNG-EYE.

This well-known Australian condition is universally considered as due to the sting of some insect. It consists of rapid and enormous swelling of the loose tissues round the eye, so that the eyelids are soon quite closed. Later the skin has a purplish hue. There is little or no pain. The swelling subsides in the course of a day or so. Cases are common in various parts of Australia, such as near Port Adelaide, near Perth, and, I believe, in the Riverina. Mr. W. W. Froggatt has called my attention to the following note on the condition, under a *nom-de-plume*, in the *Victorian Naturalist* (Vol. XIV, 1898, p. 116) :

> "While engaged botanising the other day . . . I was bitten by that terrible pest the 'blight fly,' the result being severe swelling of both eyes, suffering of pain and blindness for two or three days, with consequent loss of sleep and time while the effect of the bite lasted."

The fly seems not, as yet, ever to have been identified or even caught. Its nature, therefore, can only be guessed at. It may be one of the species of Leptidæ already mentioned, or a Culicoides.

MECHANICAL ANNOYANCE OF FLIES: CONTAMINATION OF FOODSTUFFS AND OF RAW SURFACES BY THEM.

The accounts of the early voyagers to Australia, and of explorations in the interior of Australia, contain numerous records of the annoyance caused by countless myriads of flies. Unfortunately the identification of the species causing the annoyance was not made or could not be made at the time by these various observers. In the light of our present knowledge it may be definitely stated that *Musca domestica* was not this species. It is probable that in many cases at least the agent was *Musca vetustissima.* It may be of interest to link together in this place some of the most important references that I have met with.

In 1629, Pelsart, when off the north-west coast of Western Australia, states that his boat's crew were plagued with flies, and these in such multitudes that they were scarce able to defend themselves. Dampier, who touched the north-west coast of Australia in 1688, again met with an abundance of flies, and in referring to the aboriginals, says :—

"Their eyelids are always half-closed to keep the flies out of their eyes. They are so troublesome here that no fanning will keep them from coming to one's face; and without the assistance of both hands to keep them off, they will creep into one's nostrils, and mouth too, if the lips are not shut very close; so that from their infancy, being so annoyed by these insects, they do never open their eyes as other people, and, therefore, they cannot see far unless they hold up their heads as though they were looking at somewhat over them." This reference by Dampier to the effect of flies on the eyes of the natives may possibly bear another interpretation—this is, that the disease from which the natives were really suffering was trachoma, causing scarring of the lids, &c., and thus rendering it difficult for them to open their eyes.

Flinders, when charting the north coast of Australia in 1802, again met with an abundance of flies. In November, at Prince of Wales Island, he says: "We found the common black flies excessively numerous here, and also as troublesome as Dampier describes them to be on the north coast." (Vol. 2, p. 120.) At the Pellew's Group, in December, he again refers to them: "On the shore it was hotter, yet the musketoes were not very troublesome; but the common black flies, from their impudence, were scarcely less annoying than musketoes; they got into the mouth and nose, and settled on the face or on other parts of the body, with as much unconcern as they would alight on a gum tree, nor are they driven away easily. This was the case on shore, and on board ship whilst lying at anchor, and for a day or two afterwards, but the advent of man wrought a change in the manners of even these little animals. They soon became more cautious, went off when a hand was lifted up, and in three or four days of clearing the land, they behaved themselves like other flies, and though still numerous on board, they gave little molestation." (Vol. 2, p. 173.)

At the attempt to establish a colony at Port Phillip in 1803, myriads of flies were again met with, as Lieutenant Tuckey mentions in his voyage, quoted in Bonwick's "Port Phillip Settlement," p. 25. He says: "The swamps are inhabited by myriads of mosquitoes of an extraordinary size; but the common fly, which swarms almost beyond belief, possesses all the offensive powers of the mosquito, its sting creating an equal degree of pain and inflammation." This was, of course, a biting fly, and not the common domestic fly, or *Musca vetustissima*.

Lieutenant, afterwards Sir George, Grey, also met with a similar annoyance during his exploration of Western Australia. ("Journal of Two Expeditions of Discovery in North-west and Western Australia," Vol. I, p. 81.) For instance, in December, 1837, he says, speaking of the north-west: "Sleep after sunrise was impossible, on account of the number of flies which kept buzzing about the face. To open our mouths was dangerous—in they flew, and mysteriously disappeared, to be rapidly ejected again in a violent fit of coughing; and into the eyes, when unclosed, they soon found their way, and by inserting their proboscis, and sucking, speedily made them sore; neither were the nostrils safe from their attacks, which were made simultaneously on all points, and in multitudes. This was a very troublesome annoyance, but I afterwards found it to be a very general one throughout all the unoccupied portions of Australia; although in general, the further north you go in this continent, the more intolerable does the fly nuisance become."

Captain Sturt also says ("Narrative of an Expedition to Central Australia," Vol. I, 1845–1846, p. 187), when west of the Darling, in November: "The flies were in such myriads around us that we could do nothing. It is indeed impossible for me to describe the intolerable plague they were during the whole of that day from early dawn to sunset." At Cooper's Creek, in December, he found a similar condition of things, saying that "the flies and mosquitoes were more than usually troublesome. I have not said much of these insects in the course of this narrative, for after all they are secondary objects only, as it is impossible to describe the ceaseless annoyance of these and the small ant. The latter swarmed in myriads in the creek and on the plains, and what with these little creatures at night and flies by day, we really had no rest. I continually wore a veil, or I could not have attended to our movements or performed my duties." (Vol. 2, p. 58.)

These various references indicate how abundant were the flies in the early days of discovery and exploration in Australia. Those who have had experience of the bush to-day know that the plague has in no wise decreased. In many situations in the drier parts of the interior it is almost impossible to eat one's food on account of myriads of these insects hovering around the mouth. They even continue their annoyance right into the short twilight, and it is only when darkness has finally set in do they retire to rest. Naturally, if sources of infection for the flies exist, such as uncovered fæcal material, the conveyance of organisms to food supplies could be very easily accomplished. In this way could be explained satisfactorily many of the outbreaks of typhoid fever in bush mining camps. As already indicated, the fly most commonly responsible for the above references is probably *Musca vetustissima*. Various observations of this insect will be dealt with presently.

REMARKS ON THE INDIVIDUAL SPECIES OF FLIES.

Musca domestica.

During the last few years I have had opportunities for investigating the natural history, to some extent, of this insect, and am able to formulate certain general conclusions. A review of the present habits of the fly strongly suggests that it is not an indigenous insect in Australia, but that it is an importation from abroad. This view is based upon its strong domestic traits. I find, for instance, that it prefers to associate itself more particularly with human habitations, especially houses and their neighbourhood, and that it is not found as a general rule away in the bush where houses do not exist. It is common in our houses in warm weather and in their sunny surroundings, but only occasionally accompanies an individual in his walk abroad. Similarly, if conveyed to tents and huts, it prefers remaining in their proximity. It is an insect that prefers a good light; consequently it may be excluded from houses in hot weather by keeping dark blinds pulled down. Similarly, also, it will not be found in closets which are dark, either as regards their whole structure, or the receptacle for containing the excreta. This is an important point of great value to the public hygienist. In cold weather its numbers are greatly lessened, and it may practically cease to show itself except in warm situations, such as the kitchen. It is interesting to note that its companion in houses, *Fannia canicularis*, can stand the cold better than *Musca domestica*, appearing in greater relative numbers at the beginning of and during winter than it does during summer.

The *Fannia* also may be found in darker situations than *Musca domestica*. *Musca domestica* is much attracted by collections of fæcal material in the neighbourhood of houses, and in this lies one of its greatest dangers, since, naturally, if such collection contain pathogenic bacteria, such as those of typhoid fever, these can be easily transferred on or in the bodies of flies to food materials; in fact, in various places in the world typhoid bacilli have been identified on flies from such situations. A fly thus contaminated falling into milk or milk puddings, or crawling over cold meat, may infect such foods with the organisms, and when the weather is warm considerable multiplication may occur in the foodstuffs. Flies are very frequently noticed crawling over and getting caught in jam. This led to our making investigations as to the multiplication of typhoid organisms in jam. It was found, however, that they did not multiply in this food, the strong concentration of sugar apparently soon killing them; so this may be eliminated as a vehicle of infection.

Another important aspect of this question of food contamination is that as to the minimal infective dose of typhoid bacilli. It is probable that we have from time to time ingested small quantities of typhoid organisms apparently without ill effects. It would, therefore, seem questionable whether a small dose of typhoid bacilli, conveyed, for instance, by a fly falling into a cup of lukewarm tea just before this is drunk, could have any effect upon the drinker. It seems more probable that a considerably larger dose of organisms is necessary; but this dose could be easily reached in cases where flies have fallen into milk or crawled over puddings many hours before these were eaten.

From time to time, when in the country, I have made inspections of pan closets, and have frequently found the excreta in them exposed to a more or less bright light, and *Musca domestica* in varying numbers feeding on the excreta at no great distance from the kitchen. The danger of this condition of affairs is manifest. The remedy lies in having dark closets which *Musca domestica* will not inhabit, in properly covering the excreta, or in having a mechanical device for closing the lids as exists in some of our more up-to-date towns. To obtain data as to the relative prevalence of *Musca domestica* in houses during the year I have made observations of the numbers of flies caught on two sheets of tanglefoot exposed on the breakfast table during parts of the year. The results of these observations are given in the appended tables.

A number of species of flies obtained from various situations were forwarded to Mr. E. E. Austen, of the British Museum, Natural History Branch, who has kindly identified the following. Under each identification I append some notes made as to the distribution of the species.

FAMILY, *Muscidæ*; SUB-FAMILY, *Muscinæ*.

Musca domestica, Linn.

As already mentioned, this species seems universally distributed throughout Australia in association with the habitations of man. It is naturally most abundant in the warm months, specimens being few in the winter. Its habits have already been mentioned. I have obtained it in abundance in Sydney, Melbourne, Adelaide, and Perth; in addition, the following notes have been made as to its occurrence in country districts of New South Wales :—

Milson Island, Hawkesbury River, December, 1909—

> *Musca domestica* was practically the only fly present hovering around foodstuffs during meals, but not specially annoying by attaching itself to one's person. In the open around the house it practically paid no attention to human beings.

At Bowral in April—

> *Musca domestica* was present around the dinner table. (Dr. Tidswell.)

At Bathurst at the end of January—

> *Musca domestica* was much in evidence in an outhouse attached to an hotel. They were present in a pan closet which was only moderately dark. Ashes were supplied for this pan closet, but apparently were not always used. The closet was only about 10 yards from the kitchen. The flies were in countless myriads round the stable manure adjoining the closet and kitchen. Out in the country, a few miles from Bathurst, when examining a diseased sheep with lesions on the face, several *Musca domestica* were attracted by the pus and fluids.

At Locksley, at the end of February—

> *Musca domestica* were in large numbers in the house, swarming over articles of food.

Musca vetustissima, Walk.

Walker's description of this very common species has been kindly obtained for me by Mr. W. J. Rainbow, F.L.S., of the Australian Museum. It is as follows :—

> *Musca vetustissima*, n.s., fem.—*Cana, capite albo micante, thoraci vittis duabus latis nigris, abdomine cano-cinereo, palpis nigris, antemnis piceis basi nigris, pedibus piceis, alis limpidis basi albis.* Body hoary, clothed with black hairs and bristles; head adorned with white lustre, a black stripe between the eyelets and the feelers; epistoma not prominent; sides of the face feathered with bristles at the base; eyes red, fore part flat, its facets a little longer than those elsewhere; sucker pitchy, clothed with tawny hairs; palpi black, beset with black bristles; feelers black, a little shorter than the face; third joint pitchy, linear, rounded at the top, about thrice the length of the second joint; bristle pitchy, black at the tip, about twice the length of the third joint; chest adorned with two broad black stripes; abdomen obconical, grey, with hoary reflections, a little broader,

but not longer than the chest; legs pitchy, clothed with black hairs and bristles; foot-cushions tawny; wings colourless, white at the base; wing-ribs and veins pale tawny, veins darker towards the tips; tip cross-vein forming an obtuse angle with the fourth longitudinal vein, very slightly inclined inwards along the whole length, joining the border a little above the tip of the wing; lower cross-vein nearly straight; scales white; poisers pale yellow. Length of the body 2 lines; of the wings 4 lines.

The species is at once distinguished from *Musca domestica* by the silvery white thoracic stripes. This fly is essentially an out-of-door species; very rarely indeed is it found within the house, and only occasionally in outhouses with open doors and windows. When found in these situations it is usually attracted by some source of food, such as the carcase of an animal, even though freshly killed. They have been found in abundance feeding on the pus and blood from sores on the head of sheep. As soon as one ventures outside, if the weather is warm, this species attaches itself to one's person. In some country situations, that part of one's clothing sheltered from the wind may be covered with a dense mass of these insects. They are specially annoying by hovering around and finding their way into one's eyes, nose, and mouth. It is owing to this habit that veils are so frequently worn by those persons who have to occupy both their hands. The old bushman's dodge of corks hanging round the hat, suggesting the leaden saints of Louis XI, is to keep away these flies. It is probable that these are the insects to which Dampier and many of the early voyagers refer, when mentioning the difficulty of preventing the flies being swallowed with the food.

The species can easily be obtained in the neighbourhood of Sydney, and in all the country districts of New South Wales in which I have so far been it has been met with in more or less abundance—sometimes in superabundance. It is common in Melbourne, and very common in the country round Adelaide, and is present in Perth.

Its persistence in hovering round and annoying the face of human beings distinguishes it to a great extent from *Musca domestica*, which is only slightly annoying in this respect. The abundance of the fly makes it likely that if any eye diseases, such as Epidemic Conjunctivitis and Trachoma, are transmitted by flies, this is the species to be incriminated. So far I have not met with any other species in Australia so abundant and so persistent in its attentions in this way, or sufficiently numerous to be of epidemiological import. *M. vetustissima* can quickly eat up films of blood which have been dry for days. Dried anthrax blood on a glass slide in a test tube was thus partly eaten by one of these flies, anthrax bacilli being cultivated from its excreta and the body, when emulsified and injected into a guinea-pig, producing anthrax.

Musca sp. nova.

Two or three specimens of this fly have been met with, one being caught around man in the bush, another on the carcase of a dead wallaby; both near Sydney. It has not been met with in houses. It resembles *Musca domestica* and *Musca vetustissima*, but has no black band on the dorsum of the abdomen, and is white between the eyes. It cannot be considered of any epidemiological import

Pseudopyrellia australis, Macq.

This little metallic bluey-green fly has been found hovering around fæcal material in the bush, and on the sugary manna from the grey-gum eucalyptus. It cannot be considered of importance in the spread of disease.

Fannia (*Homalomyia*) *canicularis*, Linn.

This is the well-known lesser house-fly. Some of its characteristics have already been mentioned. It is a common fly in the house, forming about ·9 per cent. of those caught on "tanglefoot" at meal-time. It appears earlier in spring than the common house-fly, apparently being able to withstand cold better. Darkness likewise has a less effect upon it as it is frequently active by gas light. I have also found it frequently in comparatively dark closets in the country. I have several times found it feeding in jars of jam. It can be distinguished from the common house-fly by its more elongated shape and by the longer wings which overlap more.

Muscina stabulans, Fln.

This fly is occasionally found in houses on the window panes, or on the table at meal-time. It is a little larger than the common house-fly. It is also found outside in the neighbourhood of houses, feeding on house-refuse, &c. Its small numbers render it of little importance. I have found it round a food-bucket as far in the country as Rowena, New South Wales.

Polietes.

Two species, perhaps new, were obtained of this genus, which have been found on window panes in houses.

Mydæa (*Spilogaster*) *addita*, Walk.

This fly has been caught on human fæces in the bush.

FAMILY, *Tachinidæ;* SUB-FAMILY, *Calliphorinæ*.

Anastellorhina augur, Fabr.

This is one of our common brownish blow-flies, being characterised by a broad blue stripe down the centre of the adbomen on the dorsal aspect. It is a widely distributed species in Australia. This fly does not seem to be attracted by foodstuffs other than meat. However, in warm weather, when meat is on the table, especially when just cooked and warm, these flies are frequently attracted inside and buzz around the table. The attraction in this case may be accentuated by the fact that the meat is not perfectly fresh, and that the cooking has increased to a considerable degree the early smell of putrefaction. They may be present around carcases even when freshly killed, but especially when undergoing changes of decomposition. They are also attracted at once by human fæces. They deposit on meat, and on fæces, young maggots, but not eggs. They have been bred from milk which has been kept and allowed to undergo decomposition in this laboratory. Around carcases of sheep in the country they are exceedingly numerous. At Locksley, in February, parts of the ground near the house were covered by large numbers of these blow-flies, which rose with a hum like the buzz of bees when disturbed. This species is apparently the *Calliphora oceaniæ* referred to by Mr. Froggatt as causing the blowing of living sheep. The frequency with which this species is attracted to human fæces in exposed situations, and the ease with which it is also attracted to meat for use for human consumption, indicates that this insect may be a means of spread of typhoid fever. If the fæces upon which it has been feeding contain typhoid bacilli, it can be easily understood that meat which is afterwards settled upon and blown may be likewise contaminated. When thus contaminated, it is probable that the meat would act as a favourable nidus for the growth of typhoid bacilli, and that when eaten some hours afterwards in a cool condition, the number of these organisms may have materially increased.

Pollenia stygia, Fabr.

This species seems to affect similar situations to the preceding, having been found on several occasions around human fæces. I have obtained it in the neighbourhood of Sydney and at Adelaide. It resembles in general the last-named species but has a golden bloom on the abdomen.

Pycnosoma varipes, Macq.

This small metallic fly has been found around dead animals at the Hawkesbury River and at Narrabeen.

Pycnosoma sp. incert.

This species has been found on a dead lamb at Rowena, and around dead animals in other places.

Calliphora sp. ? nov.

This species was found on human fæces in the bush.

Calliphora sp.

Specimens of this species have been obtained at Sydney and Adelaide.

SUB-FAMILY : *Sarcophaginæ.*

Sarcophaga misera, Walk.

This species is a fairly common one, occasionally coming inside houses. I obtained it at Adelaide and Sydney, and have found it attracted by human fæces.

SUB-FAMILY : *Dexiinæ.*

Rhynchiodexia rubricarinata, Macq.

This species in the neighbourhood of Sydney, may frequently be seen settled on rocky surfaces and fences. It somewhat resembles in general appearance a tabanid. It is of no economic importance.

FAMILY : *Piophilidæ.*

Piophila casei, Linn.

This is the common cheese mite fly. It has been obtained from cheese and from bacon.

The Flies Attracted by Human Fæces.

On December 27, 1909, some human fæces were deposited in the bush near Sydney, no houses being within several miles. Large numbers of flies almost at once made their appearance. No *Musca domestica* appeared, but a few *M. vetustissima*. There were a number of blow-flies *Anestellorhina augur* and *Pollenia stygia*. There were also a number of other unidentified species, including dark-coloured blow-flies, flies about the size of house-flies with spots on the wings, a number of minute flies, and one very large brilliant greenish blow-fly with white spots on the body.

On November 27, 1909, a dull coolish day, a sheet of tanglefoot was left in a bedroom in a suburb of Sydney for three hours over a chamber-pot containing the fæces of an infant. 20 *Musca domestica*, 4 *Fannia canicularis*, and 1 *Muscina stabulans* were caught.

Flies Round House Refuse.

The following numbers and varieties of flies were found round house refuse in a suburb of Sydney, in December : 14 *Muscina stabulans*, 12 *Musca domestica*, 5 " blue-bottles " (probably *Pycnosoma* sp.), and 3 small flies with spotted wings.

Flies and the Incidence of Typhoid Fever in Sydney.

Appended is a table giving the monthly incidence of cases of typhoid fever with the daily average for flies during the period when these cases contracted the infection. It will be seen from the figures that in *Sydney* there is no definite relationship between the incidence of typhoid fever and the relative abundance of flies. In some of the winter months, in fact, when flies are very scarce, there are as many or more cases than in some " fly " months. The opportunity given to flies in Sydney to convey infection is, owing to the prevalence of deep drainage, very small, and the cases that do occur may possibly represent those infected more directly from carrier cases employed in the preparation of food—a means which, even in winter time, may secure infection.

Infections Probably Conveyed by the Mediation of Flies.

The Barcoo Vomit or *Belyando Spue.*—That peculiar Australian condition " The Barcoo Vomit " or " Belyando Spue "—characterised by frequent attacks of vomiting during meal-time, after which the patient may return to the table and eat again, and sometimes associated with fever—has been attributed to the contamination of foodstuffs with flies. Dr. Stirling (Report of the Horn Expedition, Pt. iv, p. 132) thus describes the conditions under which the disease occurs :—

> " It is just possible, however, that actual contamination of the food by flies, if not the nauseous repugnance to it induced by their constant presence in every dish, may have something to do with it. This may seem absurd, but those who have not been in Central Australia can scarcely realise what a plague of flies really means. In less than a minute I have seen a joint of meat completely concealed from view by a covering of flies an inch thick—a moving mass—in fact, resembling a swarm of bees. A mouthful can scarcely be put into the mouth without carrying some with it, and hundreds find their way into the 'pannikins' of tea. To the horses and camels they become perfect torments, and one frequently sees the edges of the eyelids of these animals completely raw from their persistent attacks. No statement that I have made is in any degree exaggerated when the plague is at its worst, and this is soon after rain, though I admit that at other times, as indeed was the case during the greater part of the Horn Expedition, they are not nearly so troublesome. On a previous journey we encountered the plague in full force."

Dr. F. Watson (*A.M.G.*, May, 1905) also suggests the medium of flies.

Barcoo Rot.—Dr. E. C. Stirling (Int. Q. J. of Med. and Surg., Vol. I, No. 3, 1894, p. 218) suggests that this condition, which consists of superficial ulcerations very much like those of veldt sores, may be spread by flies.

Trachoma and Purulent Ophthalmia.—Both of these may be, perhaps, spread by flies. If so, *Musca vetustissima* is the probable vector.

Anthrax.—As mentioned under *M. vetustissima*, this insect may be able to spread anthrax. *Stomoxys calcitrans* may, by biting, likewise do so.

Typhoid Fever and Summer Diarrhœa of Infants.—The spread of these diseases has been attributed to flies.

Hemiptera (Bugs and Lice).

The common bed-bug *Clinorchis lectularius* is a common pest in old houses in the various States of Australia. So far there seems no definite instance of the spread of disease by this means.

Pediculosis (*Pediculus capitis*) of the head of human beings is very common amongst those who do not pay attention to the head.

Pediculus corporis and *Phthirius pubis* also occur in Australia.

Tables of Fly Captures.

The following tables illustrate well to what extent foodstuffs are liable to be contaminated by flies in the neighbourhood of Sydney. The captures were only made between some time in September of the various years and the following April or May. During the rest of the year—the cold season—though occasionally present, their numbers are so few that only occasionally is one caught.

TABLE I.—Number of Flies caught on sheets of tanglefoot on the breakfast table (8 a.m.), Neutral Bay, Sydney.

Month.	No. of Days.		Total No. of *Musca domestica.*		Av. *M. domestica* per day.		Total No. of *F. canicularis.*		Notes.
	1909.	1910.	1909.	1910	1909.	1910.	1909.	1910.	
Sept. ...	...	6	...	54	...	9	...	6	
Oct. ...	...	30	...	446	...	14·8	...	37	1 *Muscina stabulans.*
Nov ...	...	22	...	1,242	...	56	...	20	
Dec. ...	14	21	529	623	37·7	29·6	9	6	1 *M. stabulans.*
	1910.	1911.	1910.	1911.	1910.	1911.	1910.	1911.	
Jan. ...	31	19	844	559	27·2	29·4	3	5	
Feb. ...	25	7	771	83	30·8	11·8	2	...	1 *M. stabulans.*
Mar. ...	23	16	477	69	20·7	4·3	4	...	1 *Seutomyia notoscripta.*
April ...	25	3	233	2	9·3	·6	1	...	1 *M. stabulans.*
May ...	2	...	1	...	·5	...	...	...	

13 red mites were found on 7 of the above *Musca domestica*, and 1 small tick on another.

TABLE II.—Number of Flies caught as above at midday meal.

Month.	No. of Days.		Total No. of *Musca domestica.*		Av. *M. domestica* per day.		Total No. of *F. canicularis.*		Notes.
	1909.	1910.	1909.	1910.	1909.	1910.	1909.	1910.	
Sept. ...	...	1	...	10	...	10	...	3	
Oct. ...	...	10	...	167	...	16·7	...	17	
Nov. ...	2	8	46	280	23	35	...	6	
Dec. ...	11	6	411	178	37·3	29·6	5	2	
	1910.	1911.	1910.	1911.	1910.	1911.	1910.	1911.	
Jan. ...	7	7	293	305	41·9	43·6	...	...	
Feb. ...	6	...	193	...	32·1	...	...	...	
Mar. ...	5	...	134	...	26·8	...	...	...	2 cheese flies.
April ...	7	...	55	...	8	...	1	...	

9 red mites on 3 of the above *M. domestica* (6 mites were on one fly).

The following maxima of flies (*M. domestica*) have been caught at the breakfast table :—

December 28, 1909	...	109	October 17, 1910	...	81
January 2, 1910	...	189	November 7, 1910	...	142
February 23, 1910	...	126	November 10, 1910	...	192
April 10, 1910	...	83	November 11, 1910	...	134

The following maxima have been caught at the midday meal :—

January 2, 1910	...	140	February 26, 1910	...	134

TABLE III.—Table of reported cases of Typhoid Fever (Sydney Metropolitan Area), with relative daily average of flies.

(The figures for typhoid fever have been kindly supplied by Dr. W. G. Armstrong, M.O.H. for Sydney. The daily average number of flies caught on the breakfast table is that for the last half of the preceding month and the first half of the month they stand against. This is to allow for the incubation period of about half a month.)

	Cases of Typhoid Fever reported.	Daily average number of flies (*M. domestica*).		Cases of Typhoid Fever reported.	Daily average number of flies (*M. domestica*).
1909.			1910.		
September ...	35	...	September ...	53	...
October... ...	35	...	October ...	33	6·75
November ...	52	...	November ...	56	44
December ...	62	36	December ...	53	33
1910.			1911.		
January ...	96	44·5	January ...	81	25
February* ...	78	12·3	February* ...	71	38·8
March	97	38·6	March	63	5·5
April	77	13	April	44	4·3
May	75	5	May	55	...
June	68	...	June	18	...
July	57	...	July	8	...
August	69	...			

* The number of typhoid cases for February is naturally lower than in Janua and March, owing to the smaller number of days.

ON THE HÆMOLYTIC ACTION OF AUSTRALIAN SNAKE VENOMS.

By D. A. Welsh, H. G. Chapman, and E. Jean Dalyell.

(From the Laboratories of Physiology and Pathology in the University of Sydney.)

Notes were submitted of experimental work on the hæmolysis produced by the venoms of several Australian snakes when brought into contact with the red blood cells of various animals. This interaction was tested with graded amounts of venom (unheated and heated) in the presence of (*a*) the washed corpuscles alone, and (*b*) the washed corpuscles together with homologous or heterologous blood serum (unheated and heated). The phenomena of venom-hæmolysis were compared and contrasted with those of serum-hæmolysis. The authors regret that, owing to pressure of other work, they were unable to have their paper ready for publication in the Proceedings of Congress. Some earlier observations were published in the *Australasian Medical Gazette*, July, 1910.

Experimental Carcinoma and Sarcoma in Mice and Rats.

A demonstration was given by Professor D. A. Welsh of some experimental work on carcinoma and sarcoma that he had carried out in the laboratories of the Imperial Cancer Research Fund under the direction of Dr. E. F. Bashford. A series of thirty-three specimens, prepared in the above laboratories to illustrate experimental new growths in mice and rats, was exhibited in the Congress Museum.

A PRELIMINARY REPORT ON THE BACTERIOLOGY OF THE BLOOD.

By Mary B. Burfitt, B.Sc., M.B., C.M. (Syd.).

Resident Pathologist, Royal Prince Alfred Hospital.

Since February of this year I have made 138 blood cultures from 128 patients, more than one culture having been taken in some instances from the same case. The following results were obtained:—Typhoid bacillus, 23; Paratyphoid bacillus, 3; Streptococcus, 13; Staphylococcus pyogrenes aureus, 3 times from the same case; a diplococcus from a case of rheumatism; a doubtful pneumococcus; a doubtful gonococcus; and a colon bacillus.

Methods.—In making the cultures 10 c.c. of blood were, as a rule, taken from one of the superficial veins at the elbow.

Media.—In cases of suspected typhoid, ox bile, filtered, sterilised and undiluted, was generally employed; in other conditions, bouillon. However, it seems immaterial what medium is employed, as I have found both the typhoid bacillus and streptococcus to grow equally well in both.

Dilution.—In the earlier cultures 10 c.c. of blood were put in about 30 to 40 c.c. of bouillon or 30 c.c. of bile. On one occasion there were in hospital two patients who clinically appeared to have septicæmia. Cultures were made in the usual way and proved sterile. It was suggested that perhaps the dilution was unsuitable, that there might possibly be some anti-body in the serum inimical to the growth of the bacteria. Fresh cultures were made four days after the previous culture that had proved sterile in each case. 10 c.c. of blood were put in 500 c.c. of bouillon in the one case, and into 100 c.c. in the other. From each a streptococcus was cultured. To ascertain if the concentration of the serum was the cause of failure in the first instance, a series of dilutions were tried in some subsequent cultures. A 20 c.c. syringe was used. About 4 c.c. 2 per cent. sodium citrate were drawn up into the syringe, the vein punctured, and 16 c.c. of blood withdrawn. The citrate and blood were well mixed, and 5 c.c. of the mixture put into 5 c.c., 10 c.c., 25c.c., and 50 c.c. of bouillon. In this series two growths of a streptococcus and one of the typhoid bacillus have been obtained, the organisms growing equally well in all the dilutions used. I hope to obtain more results in this series, but at present the results go to show that the dilution is of little consequence.

After from twelve to eighteen hours' incubation, the first sub-culture is made on an agar slope, and if this should be negative, repeated sub-cultures are made at intervals of twenty-four hours, until the fourth day, when if there is still no growth, the culture is regarded as negative.

Results.—Enteric Fever : In the *Australian Medical Gazette,* April, 1911, is an article by Dr. Hamilton Tebbutt on the bacteriology of typhoid fever. I have followed up his method of investigation and the results support his contention that in certain cases of enteric the blood culture method is a surer means, and a means of earlier diagnosis, than is afforded by Widal's test.

Cultures were taken from 38 cases of enteric; 23 of these, or 60 per cent., gave positive results. Table I gives a list of the cases from which positive cultures were obtained, and also the 3 cases from which a para-typhoid bacillus was cultured. Each bacillus was identified by its bio-chemical reactions in a series of peptone solutions containing sugars, &c., as given in the table, where "A" indicates acidity, and "G" the formation of gas in a Durham's tube.

The cases from which the bacillus was not obtained are cited in Table II.

The average day on which the bacillus was found in the blood was the eleventh, though it was found as early as the third day, and as late as the fifth week. The latter patient died. Generally those cases in which the bacilli were still present at a late stage of the disease were the more severe. The average day on which negative results were obtained was fourteenth. In 26 per cent. of the cases giving a culture, a positive diagnosis could be made, while the Widal reaction was still negative.

TABLE I.

Case.	Day of disease Culture obtained.	Day of disease Widal tested, and result.	Litmus milk.	Glucose.	Mannite.	Dulcite.	Lactose.	Cane sugar.	Arabinose.	Raffinose.	Sorbite.	Indol.	Day of disease after which temperature normal.	
1	6th	6th +ve	A	A	A	O	O	O	O	O	A	O	35th	
2	10th	11th —ve 16th —ve 22nd +ve	A	A	A	O	O	O	O	O	A	*	18th	* Not tested.
3	5th	5th —ve 9th —ve 11th —ve 18th +ve	A	A	A	O	O	O	*	*	*	*	18th	* Not tested.
4	13th	13th +ve	A later alk.	A	A	O	O	O	O	O	A	O	29th	Temperature intermittent.
5	10th	10th +ve	A	A	A	O	O	O	*	*	*	O	26th	* Not tested.
6	10th	10th +ve	A	A	A	O	O	O	O	O	A	O	30th	Very severe toxæmia.
7	20th	20th —ve 23rd +ve	A	A	A	O	O	O	O	O	A	O	...	Severe case, lasting 43 days, followed by relapse of 17 days.
8	12th	12th +ve	A	A	A	O	O	O	O	O	A	O	31st	
9	11th	11th +ve	A	A	A	O	O	O	O	O	A	O	37th	Very severe toxæmia.
10	15th	15th +ve	A	A	A	O	O	O	O	O	A	O	43rd	
11	5th	5th +ve	A	A	A	O	O	O	O	O	A	Tr.	17th	Colon bacillus infection of middle ear.
12	11th	11th +ve	A	A	A	O	O	O	O	O	A	O	16th	Very mild case.
13	(?) 22nd	(?) 22nd +ve	A	A	A	O	O	O	O	O	A	O	(?) 35th	Illness dated from miscarriage.
14	14th	14th +ve	A	A	A	O	O	O	O	O	A	O	32nd	
15	15th	15th +ve	A	A	A	O	O	O	O	O	A	O	18th	Relapse 33rd day for 5 days.
16	9th	9th +ve	A	A	A	O	O	O	O	O	A	O	31st	
17	(?) 8th	(?) 8th +ve	A	A	A	O	O	O	O	O	A	O	(?) 18th	
18	3rd	3rd +ve	A	A	A	O	O	O	O	O	A	O	12th	Onset with rigor.
19	7th	13th +ve	A	A	A	O	O	O	O	O	A	O	23rd	Relapse 28th day for 14 days.
20	6th	6th —ve 9th —ve	A	A	A	O	O	O	O	O	A	O	32nd	Agglutination not tested after 9th day.
21	(?) 5th week.	(?) 5th week —ve	A	A	A	O	O	O	O	O	A	O	...	Died from hæmorrhage and toxæmia.
22	19th	19th +ve	A	A	A	O	O	O	O	O	A	O	...	
23	6th	7th —ve 11th +ve	A	A	A	O	O	O	O	O	A	O	...	
24	6th	...	A	AG	AG	AG	O	O	*	*	*	O	26th	Very severe toxæmia.
25	7th	...	A	AG	AG	AG	O	O	*	*	*	O	20th	
26	8th	...	A	AG	AG	AG	O	O	*	*	*	O	19th	

TABLE II.

Case.	Day of disease Culture, negative.	Day of disease Widal tested, and result.	Day of disease after which temperature normal.	
1	9th	9th — ve	44th	Had also colon bacillus pyelitis.
	13th	12th — ve		
		23rd + ve		
2	11th	14th — ve		
	14th	26th — ve	24th	
		28th + ve		
3	16th	16th + ve	37th	
4	11th	11th + ve	15th	Two slight recrudescences 23rd and 37th day of disease.
5	8th	8th — ve	40th	Relapse 63rd day for 21 days.
	65th	11th + ve		
6	10th	10th + ve	13th	Relapse 37th day for 9 days.
	13th			
7	14th	14th + ve	24th	
8	15th	15th + ve	20th	
9	16th	16th + ve	20th	
10	16th	16th + ve	25th	
11	15th	15th + ve	......	Mild remittent and intermittent temperature for 40 days.
12	4th week	4th week + ve	......	Normal temperature 3 days after Widal tested.
13	5th week	5th week + ve	......	Intermittent temperature for 40 days.
14	18th	18th — ve	26th	Relapse 40th day for 5 days.
		23rd + ve		
15	11th	11th — ve	18th	Recrudescence 24th day for 5 days.
		13th + ve		

Paratyphoid Fever.—From three cases a bacillus corresponding to the paratyphoid α of Schottmüller was grown. It was an extremely motile bacillus, caused permanent acidity in litmus milk, and acid and gas production in glucose, mannite, and dulcite. There was no formation of indol. Agglutination tests were as follows :—

Case.	Widal.							
	Period of pyrexia.				4 months later.			
	< Stock Ty. Culture.		< Autogenous Culture.		< Stock Ty. Culture.		Autogenous Culture.	
	1 in 60.	1 in 120.	1 in 60.	1 in 120.	1 in 60.	1 in 120.	1 in 60.	1 in 120.
24	...	......	...	...	—	—	+	+
25	+	Incomplete clumping.	+	+	—	—	—	—
26	+	—	+	+				

These cases clinically resembled enteric. 25 and 26 were mild, but 24 was an extremely severe one.

Streptococcal septicæmia.—Of this, there were thirteen cases. Writing of streptococcal septicæmia, Osler says : " We are beginning to recognise the fact that these infections are not always so serious as we thought. Death may occur within twenty-four hours, or be delayed for several days, even for weeks, and recovery may occur." Of the patients from whose blood I cultured streptococci, 9 died, 3 recovered, and 1 is at present in hospital. The cases that proved fatal did so after periods varying from four days to fifty-four days from the onset of the disease. The recoveries occurred after sixty-three days, twenty-five days, and ten days pyrexia. These cases therefore illustrate how severe and how comparatively mild may be such a blood infection.

The patient from whose blood the staphylococcus pyogenes aureus was cultured died. *Post mortem* a large subphrenic abscess was disclosed.

Acute Rheumatism.—Cultures were made from six patients, during periods of pyrexia, with only one positive finding. This latter was from a girl 9 years of age, with articular and cardiac lesions. Previous attack two years before followed on scarlet fever. At the time of taking the culture her temperature was 100°, pulse 120, and R. 50. She had also some pneumonic signs.

The organism cultured was a gram + ve diplococcus. On agar the growth resembled that of the streptococcus, but was slightly more vigorous. In bouillon it formed a deposit at the bottom of the tube, but this did not have the sandy appearance characteristic of the streptococcus. Films made from the culture on agar, and from the bouillon, showed diplococcal forms, with a few short chains of from six to eight cocci. An emulsion of the culture was injected into the peritoneal cavity of a rabbit. About forty-eight hours after the injection the rabbit appeared seedy, and died six days later. *Post mortem* nothing was disclosed other than some whitish areas in the liver, which microscopically proved to be areas of necrosis. Cultures made from these and from the heart blood and joints were sterile.

Rheumatoid Arthritis.—Four cultures were made, two of them during periods of pyrexia. All proved sterile.

Pneumonia.—Cultures were made from ten patients, at periods of the disease varying from the fourth to fourteenth day. One culture was obtained of an organism culturally resembling a pneumococcus, but on injection into a rabbit failed to cause death.

Gonorrhœa.—Cultures were taken from two patients with acute gonorrhœa. From one of these it was taken within twenty-four hours of his having had a rigor, and a diplococcus, which culturally resembled the gonococcus, was obtained. The staining of the cultures was indefinite, some of the diplococci being gram negative, others gram positive. The culture injected into a rabbit produced no effect. The other culture was negative.

The remaining positive culture was from a man who had several rigors after the passing of a catheter. In this case a gram negative nonmotile bacillus was cultured. It produced slight acidity in litmus milk, and acid, and gas in glucose. This was an interesting case, inasmuch as it offers a possible explanation of the so far unexplained catheter fever. I have not yet had an opportunity of attempting more than one other culture from such a case, and this one was negative.

Apart from the question of specific diagnosis, the practical importance of these cultures lies in the fact that in positive cases an autogenous vaccine can be prepared and administered to the patient within thirty hours of the taking of the blood, and in many of these cases this was done.

In conclusion, I wish to express my thanks to Professor Welsh for many suggestions and references.

NOTES ON THE EFFECTS CAUSED BY THE BITE OF A RED-BACKED SPIDER (*Latrodectus liasseltic*, Thor.).

Dr. John Wilson Sutherland, Narromine, N.S.W.

(Communicated by Walter W. Froggatt.)

When Dr. Sutherland told me he had been badly bitten by a red-backed spider at Narromine, I asked him to put the symptoms down in writing for me. He writes thus, also sending me a specimen of this well-known red-backed spider: "*Re* these red-backed spiders, I may say that they abound in this neighbourhood, and scarcely a latrine is free from them; consequently, the common site of the bite on man is on the penis or scrotum. I myself was bitten on the anus. As regards symptoms, there was first of all the actual bite, which resembled the burn from a red-hot needle. This was succeeded in a few minutes by a burning pain all round the bite. The comparison I can give is that it resembled the sensation experienced after a severe stinging of nettles. This persisted for forty-eight hours. At no time had I any of the symptoms of collapse or shock, nothing but this constant 'nettle-sting' pain. After forty-eight hours the burning was succeeded by severe itching. I had a curious sequel in the shape of neuralgia in the soles of my feet, with sweating. This lasted for two days more. What interested me most was the occurrence of plantar nerve neuralgia, no tenderness along the nerves, nothing but frequently occurring pains in the soles of my feet.

I saw the spider that bit me. It was one of those ones which have a vermilion streak antero-posteriorly in the centre of the back. I will send some in a day or two. Some of the victims here are said to have been delirious for hours. Intense pain is the common symptom. Profuse sweating occurred in very many instances. Ecchymosis and œdema in the area around about the wound are also not infrequent. Tobacco juice from the pipe is a common remedy; and I know one man who was bitten on the arm, had the wound freely incised, and this tobacco juice, or as he called it 'nicotine,' rubbed in, and had absolutely no evil effects, although the spider was a large one. I applied tincture of iodine, and then 1 in 20 acid carbolic solution, and am sure the frequent application of the latter did much to prevent more serious effects. Frequent rubbing with a menthol cone did much to ease the burning sensation."

Another Case of Spider Bite.

I mentioned in a former note, that I had met with another case of spider bite. My patient, a very healthy young man, felt something bite his leg one evening, and soon after began to suffer from a severe pain in the testicle on the same side. Shortly afterwards the pain shifted to his back, and became almost unbearable. I saw him about 2 a.m. His abdominal muscles were firmly contracted and he complained of pain in his back. The temperature and pulse normal; on the calf of the right leg was a small wheal where the bite had been. No other symptoms or signs could be made out. A hypodermic injection of morphine gave relief. The curious state of contraction of the abdominal muscles persisted for a day or two, and the pain was still felt in a less degree for a day or two.

VI.

SECTION OF PUBLIC HEALTH AND STATE MEDICINE.

PRESIDENT'S ADDRESS.

By J. S. Purdy, M.D., C.M. (Aber.), D.P.H. (Camb.), F.R.S.,E., F.R.G., S.
Chief Health Officer for Tasmania.

Conditions of Work and Health.

It is usual for the President of the section of a Congress such as this either to acknowledge the honour that has been conferred upon him or to apologise for inflicting an address on his audience. Whilst conscious to myself that the honour is not so much to a little man as to the little State he has the honour to represent in my present, to me unprecedented, position, I may claim at least the clemency usually allotted to a first offender. In choosing to discuss questions of Industrial Hygiene, or Social Medicine, I have been guided to some extent by the fact that, in addition to attempting to fulfil the duties of Chief Health and Quarantine Officer for Tasmania, it has recently fallen to my lot to occupy the somewhat unenviable position of the first Chief Inspector of Factories and Administrator of the Wages Boards Act in that State.

Without wishing to enter into autobiographical details, I may mention that during the twenty-one years I have been concerned with the study and practice of medicine my work has brought me into close touch with the conditions under which people live and work in different parts of the world. Since taking up the special work of a sanitarian some ten years ago, a fortuitous combination of circumstances has given me the opportunity of public health service in six countries—South Africa, England, Egypt, the Sinai Peninsula, New Zealand, and Tasmania.

Although each country has certain individual characteristics as to people, climate, and conditions, yet there are some local outstanding features which are common to all. Thus it is a matter of common observation that the man who lives an outdoor life is, generally speaking, more robust and healthier than the individual who is condemned voluntarily or involuntarily to follow an indoor or sedentary occupation.

All employment may be roughly divided into out-of-door and indoor—dusty and non-dusty.

Whilst indoor occupation, broadly speaking, is inimical to health unless carried on under ideal conditions, outdoor work in a pure air favours health and longevity.

Out-door Occupation.

Every student of public health is acquainted with the famous occupational mortality tables of Dr. Tatham, compiled for the Registrar-General for England. Few people, however, have yet imbibed the wholesome lesson which they teach, which is that the more natural the environment and the less artificial the conditions of life of the worker the longer his days in the land. Thus, farmers are shown to be thrice as healthy as labourers in industrial centres. Rohe, in his "Text-book on Hygiene," shows that farmers and gardeners have the greatest expectation of life in the State of Massachusetts, with an average of 65·29 years, whilst females engaged in wage-earning occupations have an average of 39·13 years.

It was only comparatively recently that investigation had been made as to morbidity and mortality in different occupations. As a matter of actual fact, it was impossible in Australia to-day to get such data extending over any considerable period of time. Thanks to the initiative of the Commonwealth Statistician in introducing uniformity in the recording and compiling of death returns, it will ultimately be possible in Australia to get occupational mortality tables showing the effect of occupations at different age-periods on the general health.

It is interesting to know that from the national economic standpoint the healthiest industries, agricultural and pastoral, have always been the most profitable at all times and in all countries. Thus, under whatever dynasty you study Egypt, whether the mainspring of good government be a Joseph or a Cromer, what Charles Rollin, the French historian, wrote over 200 years ago in "The Manners and Customs of the Egyptians," is that "Egypt owed its riches and plenty to husbandmen and shepherds." It will be always so with every kingdom where governors direct all their actions to the public welfare. The culture of lands and the breeding of cattle will be an inexhaustible fund of wealth in all countries, where, as in Egpyt, these profitable callings are suggested and encouraged by maxims of State policy.

Again, the Abbé Fleury, in his work "Of the Manners of the Israelites," states:—"Whatever artifice or craft may be used to convert money into commodities, and these back again into money, all must ultimately be owned and received from the products of the earth, and the animals that it sustains and nourishes. Let us imagine a country where sloth, effeminacy, and the ignorance of things necessary for life are held in just contempt, and where pleasure is less valued than health and bodily strength; in such a country, it will be much more to a man's reputation to plough and keep flocks than to waste all his hours in sauntering from place to place, in gaming and expensive diversions." But we need not have recourse to Plato's Commonwealth, or even that of Australia or the Dominion of New Zealand, one might add, for instances of men who have led these useful lives. It was thus that the greatest part of mankind lived during near 4,000 years, and that not only the Israelites, but the Egyptians, the Greeks, and the Romans—that is to say, nations the most civilised and most renowned for arms and wisdom. They all inculcate the regard which ought to be paid to agriculture and the breeding of cattle.

It is being universally recognised, therefore, that the prosperity of a people and country depends largely on agriculture, and that those who are employed on the land enjoy, in the main, the best health, which is granted to be the best asset men and a country can have. One wonders why agriculture is not taught together with hygiene in every school to every boy and girl in Australia and New Zealand. It is a matter of actual observation that the earliest years of life are the most impressionable.

Everyone knows the old Jesuitic saying, so often quoted nowadays by advocates of the Kindergarten, "Give me a child until he is 7, and I do not care who has him afterwards."

Therefore, to attempt in some measure to counteract the unfortunate tendency people have to seek a gregarious existence, causing them even in our now largely undeveloped countries to gravitate towards the towns, every child that is born into this Australasian world alive—be he either a little liberal or a little conservative, city or country bred—should be taught by precept and example to take an interest in the growth of plant-life, the tilling of the soil, and in agricultural pursuits generally.

There is much need of the teacher of agriculture and hygiene who has himself been taught, and who practically applies his teaching.

We hear from time to time an outcry against child-slavery on farms. Undoubtedly it is a curse, and requires to be brought under rigid control. At the same time, the earlier the age at which a child learns to milk and make himself useful about a farm the more likely is he in after life to develop the habit of work, to stick to the land, and become a useful, practical farmer.

What Australia wants, what New Zealand wants, we are told, is immigrants. We certainly have plenty of room for more people. My limited experience of immigrants, however, is such as to cause me to remark, "Get them young, not after they have formed habits and become adapted to artificial conditions of life such as are associated with dwelling in big cities and congested centres of population."

It might be worth while getting a few thousand of Baden Powell's picked boy scouts from the old land, and distributing them about the country. It is only a truism to state that the best immigrants for any country are children born in the country. Assisted passages are given to other people to come here from outside; therefore one welcomes the movement to give assistance to Australian and New Zealand motherhood. Not only from an economic but also from a public health standpoint, and for the future of the race, every encouragement should be given to people to go on the land, to marry, and to rear a family. Such a policy means increased prosperity and improved conditions of health for the people. As to the individual profit, to quote Lord Francis Bacon, "The improvement of the ground is the most natural obtaining of riches; but it is slow; and yet, when men of great wealth do stoop to husbandry it multiplieth riches exceedingly."

Australia and New Zealand, although in the history of countries of comparatively mushroom growth, yet can give abundant evidence of the accumulation of wealth by the development of agricultural and pastoral industries.

Although we know that the country-bred man is, man for man, physically a better man than the townsman, and enjoys more robust health, generally speaking, yet we, who in some measure have the guidance of the public health conscience of the people, cannot but be ashamed of some of the conditions under which people live and work in the country.

I know that I had to stand, quite deservedly, a little chaff from some of the Scottish Farmers' Commissioners, who commented on some of the primitive sanitary, or rather insanitary, arrangements or lack of arrangements of some of the farms they had visited. Knowing, however, the conditions of some rural districts in England and Scotland, one was able to remark, "People in glass houses should not throw stones." However, one trusts that when the teaching of hygiene in schools becomes general we will ultimately get a generation giving us a public and members of local authorities who will put sanitation, with a capital "S," in the forefront of their local parochial programmes of reform, and not relegate it to a position of less importance than the provision of roads and bridges or the registration of dogs.

Unfortunately, in the Commonwealth the proportion of people engaged in industries which are not strictly agricultural or pastoral, when one considers the vast area of land, is comparatively high. To quote from the Commonwealth Year Book:—"A feature of the distribution of population in Australia is the tendency to accumulate in the capital cities. To such an extent is this metropolitan aggregation carried, that in every State the population of the capital far outnumbers that of any other town therein, and ranges between 20 and 44 per cent. of the entire population of the State." Thus, even in Tasmania we have one-third of the entire population congregated in the two cities and suburbs of Hobart and Launceston.

The English Royal Commission in 1904, whilst unable to prove the existence of actual deterioration, there being no past standard with which the people could be compared, published much valuable evidence to show that certain sections of the people, more especially in the more congested areas of cities, were liable to suffer from the ill effects of an artificial, unnatural environment.

In Australia and New Zealand, although the conditions under which the masses live are vastly better than in the Old Country, still it is comforting to read the statement of the late Professor Cunningham, the distinguished anthropologist:—"In spite of the marked variations which are seen in the physique of the different classes, anthropologists believe, with good reason, that there is a mean physical standard, which is the inheritance of the people as a whole, and that no matter how far certain sections of the people may deviate from this by deterioration, the tendency of the race, as a whole, will always be to maintain the inherited mean. In other words, those inferior bodily characteristics which are the result of poverty (and not vice, such as syphilis and alcoholism), and which are therefore acquired during the lifetime of the individual, are not transmissible from one generation to another. To restore, therefore, the classes in which this inferiority exists to the mean standard of national physique, all that is required is to improve the conditions of living, and in one or more generations all the ground that has been lost will be recovered."

Together with the sister oversea Dominions of Canada and South Africa, Australia and New Zealand offer a field for studying the effects of what is or ought to be generally a healthy and natural environment on a people, for the most part at least, only one or more generations removed from the present stock of the British Isles, or in the case of parts of Canada and South Africa, people from France and Holland. Already sufficient data has been compiled with regard to school children at least to show that the improved environment has had a beneficial effect. Records collected by Mr. Symons and myself for the New Zealand Health Department, quite as extensive as to numbers examined as the data available to witnesses at the time of the royal commission, were especially interesting in showing that whilst the average stature at the ages of 11 to 12 of boys in the public schools in England in 1883 was 54·98 inches, that of the cadets of the Auckland schools and King's College was 56 inches at the same age. Contrasting the heights of boys at Marlborough College, England, with those of King's College, Auckland, and Wanganui College, we found between the ages of 14 and 15 the figures were 61·96 for the famous English public school, and 63 for the New Zealand schools. Even more interesting was the fact that a comparison between the boys of the Chapel-street school, Auckland (probably the children of the poorest parents of the largest New Zealand city), with the boys of Remuera (the wealthiest suburb) showed less disparity than has been found to exist between the measurements and weights of children in the poorer districts of English cities as compared with the children of the more prosperous parents resident in the suburban centres and the boys of the English public schools. Such a result was only to be expected in a country where there is not the severe handicap of poverty, which is such a dead-weight in some of the congested centres of the Old Country. The prowess of the young Australian and New Zealander in the field of war or of sport is also such as need not make him ashamed to meet the stranger either within or without his gates.

The fact that already in the Commonwealth of Australia, with a population of less than 5,000,000, there are over a quarter of a million persons employed in factories, shows that already we have our industrial problems, some of which have to do with industrial diseases.

Indoor Occupation.

Persons habitually engaged in hard work indoors, and especially in factories, are more liable to contract certain diseases, suffer from a greater amount of sickness, and have not the same average expectation of life as those working out of doors. Without minimising the influence of insanitary dwellings, improper and insufficient food, lack of recreation, possible indulgence in alcohol and other factors, there is no doubt that the chief danger of indoor life is exposure to vitiated or respired air. The air of dwellings and factories is never so pure as the outer air, owing to pollution from the products of respiration, combustion, and decomposition. The mere conglomeration of individuals also tends to vitiate the air with dust, germs, and organic matter from the skin, mouth, lungs, and soiled clothing.

The character of the occupation, more especially the production of dust and the nature of the dust, has also even more to do with the industry from a health point of view. As early as the first half-century after the birth of Christ we find Pliny, the Roman historian, writing of certain diseases which were peculiarly the diseases of slaves, viz., lead and quicksilver poisoning, the consumption of pottery workers and those engaged in the textile industries of that time. Although some seventeen and a half centuries later, in the British Empire, owing to the development of a more humanitarian spirit, we abolished slavery—not without a bitter and hardly-fought fight—yet we still have Pliny's diseases of slaves, only now we call them "industrial diseases."

Quicksilver-poisoning is to-day a negligible factor, but lead-poisoning, although getting yearly less, is, like the poor, still with us. Consumption still claimed thrice as many victims from those working indoors as from those engaged in the more natural open-air employments, the chief of which was the cultivation of the soil.

Occupations involving exposure to irritating dust.

Two groups of industries have been recognised as especially harmful unless carried on under rigid control and hygienic conditions. In some trades this was an almost impossible ideal, viz.:—(1) Trades which give rise to mechanical or chemical irritation of the lungs by dusts or vapours; (2) trades in which the workers were exposed to great varieties of temperature.

Among those engaged in occupations usually classed as dangerous to health are bleachers, bookbinders, brassfounders, compositors, electrotypers, stone-cutters, gasworks employees, white-lead workers, persons employed in the manufacture of explosives, firemen, potters, file-makers, and rubber-factory workers. The danger and death rates in certain trades are directly proportional, not so much to the amount of dust produced as to the character of the particles which compose it, and the conditions under which the work is carried on.

According to Summerfield, the mortality in Berlin of persons engaged in non-dusty occupations is 2·89 per 1,000, whilst for persons engaged in dusty occupations it is 5·43 per 1,000. The mortality of the Berlin population at the same age is 4·93 per 1,000.

It has long been recognised that the sharp, angular, gritty particles of iron, quartz, and stone-dust are more liable to produce injury of the lungs than coal, flour, wood, grain, and other kinds of dust.

Bronchitis, pneumonia, and tuberculosis are notoriously prevalent in certain industries where dry dust is produced, as among the rock-choppers of Sydney, the quartz-miners of Bendigo, Reefton, and Mathinna.

Other dusts, such as the innocuous bland coal-dust, instead of being really harmful, might be beneficial, more especially in lessening the rate of consumption. As a matter of fact, as Dr. Trotter, of Bedlington, and myself pointed out in the *British Medical Journal* in 1903, in the North of England coal-mining centres the death-rate from phthisis was higher among women than among men, owing to the bad housing of the people, women spending more time indoors.

It is remarkable that miners, who, as a class, are generally so insistent on reform, have so long remained tolerant of the obviously unsatisfactory conditions under which they dwell. Here one may mention that the advances to workers in New Zealand, by which the wage-earners can build houses for themselves, is a measure which is already tending to improve the health of the people.

The death-rate from consumption among Cornish miners was treble that among coal-miners.

Other metallic and mineral dusts.

In the cutlery and tool industry, especially in the grinding and polishing departments, there is the dual danger of dust from the metal and the grindstone or emery wheels. Beyer found that of 196 needle-polishers at Remischird, only twenty-four were over 40 years of age. In this occupation, unfortunately, unlike some of the others, the "wet process" cannot be employed, as the small objects, such as needles, have to be brought close to the eyes, thus increasing the inhalation of metallic dust. In this industry, as in most other dangerous industries, the greatest obstacle to improving conditions is unfortunately the operatives themselves. Thus, to quote from the 1909 Massachusetts State Board of Health Report:—"Even when employers have provided hoods, connected with a system of exhaust fans or blowers, a very large proportion of grinders recklessly remove the hoods, and thus expose themselves unnecessarily to this especially dangerous form of dust. They assert that they prefer freedom of movement, with dust, to the protection offered by hoods."

Thus, as Professor Koch foretold in 1883, that it would be necessary to wait for another generation of medical men before the fact that consumption was a communicable disease, and could be prevented, was universally recognised, so it will be necessary to wait for another generation of quartz-miners and needle-grinders before the prevention of the mortality of dust-inhaling diseases can be secured by the practice of reasonable precautions.

Probably the lead trade was the most dangerous of all industrial occupations. According to a French writer, there were 111 industries in which this metal was handled. In England, which, as in the inauguration of most sanitary reforms, has led the way, reliable data are obtainable as to the number of cases of lead-poisoning.

The German Industrial Insurance Institutes also furnish not only the number of deaths, but also the number of cases treated, together with the age period and the duration of the disease. Great differences are found in the conditions under which the work is performed, some of which are avoidable. Thus, it is claimed to be unfair to characterise certain trades as dangerous when experience has shown that no harm results when proper safeguards have been taken. There are, for instance, a number of occupations in which the alcohol habit prevails, maybe because of the character of the work or as a result of association; thus it is not always fair to attribute ill-health of the workers to the character of the employment.

The making and setting of type, the manufacture of paint, glazing pottery, and making yellow dyes were the chief branches of the more dangerous trades working with white lead. In England women had been prohibited from employment in the more dangerous lead processes, as they were more susceptible to lead-poisoning than men. In Newcastle-on-Tyne, before the new factory regulations dealing with lead workers were introduced, Sir Thomas Olliver showed that there were ninety-four cases of lead-poisoning among women as against forty-one men.

Phosphorus-poisoning will soon disappear, as the prohibition of the manufacture and sale of white phosphorus matches becomes general. France made match-making a Government monopoly, but owing to the large sums paid in compensation to the phosphorus-poisoned workers she was forced to find a non-poisonous salt. In well-ventilated, clean factories the making of matches is no longer a dangerous occupation.

There were many industries in which men and women were employed which were not in themselves dangerous, but unfortunately became so owing to the unhealthy conditions under which they were sometimes conducted. Thus there ought to be nothing really harmful in tailoring, dressmaking, the occupation of saleswomen, shop-girls, or domestic service; yet in the past some of these trades and employments had been conducted with so little attention to the provision of sufficient cubic space, good lighting, hours of employment, ventilation, provision of lavatories for washing, and even sanitary conveniences that the health of some women had been prematurely broken down.

Long hours, dirty rooms, and overcrowding were not only not essential to an indoor industry, but were also uneconomic, as it had been proved that the provision of good, airy workrooms had increased production, more regular attendance of employees, with less absence from sickness. It would be impossible to exaggerate the ill-effects of close application to work indoors upon women's health. Women were not physically adapted to undergo great strain and endurance at work. This was well illustrated by occurrence of flat feet so frequent in some overworked hospital nurses and shop-girls, as well as the occurrence of varicose veins, the result of the strain of standing on their feet for hours, combined with the effects of constipation and anæmia.

The nervous strain on young women incidental to watching machinery and keeping up with its speed broke down a woman more than did the old-fashioned spinning and weaving with the foot-power looms. Bodily fatigue was the precursor of illness. Cravings for tea or other stimulants, constant headache and backache, often were the result of girls working in ill-ventilated factories and shops. It was now universally recognised that it was inimical to national physique for married women and prospective mothers to work in factories. Legislation was now almost universal to prevent women working right up to the time of confinement or some time afterwards.

In England from 40 to 50 per cent. of the babies born to factory mothers died in infancy.

Now, we must remember that there had been a great awakening to the importance of this subject, largely the result of the revolution in the industrial world. In spite of the inevitable mistakes which may have been, and might be, committed by trades-unionism, still this had been the great force in our time for improving the conditions of the workers. The introduction of machinery last century made impossible the retention of the survival of the remnants of the old feudal system of labour, when the employer was himself a master craftsman, and worked at the bench with his apprentices and journeymen. The master became the owner of a factory, the workmen were pieces in a machine. The employer became more a brain-worker, and at first the employee more mechanical and less interested in the ultimate result of constructive work, owing to specialisation in manufacture. It became harder for the employee to become an employer owing to the expense of starting a factory. Women and children could serve machines, and cost less than men. The ditch between master and man gradually widened, until it became a gulf. The combination of the workmen, the formation of benefit societies, and the agitation for better conditions, combined with the extension of the franchise, brought reform.

Now the pendulum was swinging back towards bridging the gulf, and co-operation between employer and employee was not only advocated, but had become practically essential to the carrying on of industries. It had been necessary for the State to ensure better conditions for the worker, and all classes were realising the necessity of working for the common good. When I remember the conditions among the Northumbrian miners when I used to play football against them, and afterwards as I worked among them as a colliery surgeon for a short time, and contrasted them with the conditions existent at the time the Right Hon. Thomas Burt, the member for Morpeth and Father of the English House of Commons, was working as a boy in the mines, and the conditions of to-day, when they are actually considering the introduction of change-houses and baths, I realise that some twenty or thirty years hence, owing to the Town Planning Bill and other more recent legislation, the old conditions of overcrowding, dirty, midden streets, lack of ventilation and lighting, would pass away, as it was reasonable to expect an even further advance in sanitation than in the past.

It is gratifying to realise that the conditions which gave rise in the past to so much industrial disease in the older countries can never now be reproduced in Australia and New Zealand. At the same time, the workers must themselves assist in all efforts made to improve their environment. It is useless putting sanitary conveniences into mines, or baths in change-houses, if the men, as at present, when they are provided, either persist in not using them or in some cases abusing them.

Probably it will not be until we get a generation of workers educated in simple rules of health, and accustomed to discipline and well-regulated habits, that much of the sickness still associated with indoor work will be lessened.

It is pleasing to reflect that since the appointment of a member of our profession as Chief Inspector of Factories in England, Dr. Whitelegge, and later the appointment of Dr. T. M. Legge, and more recently Dr. Collis, there has been a great advance in the application of sanitary knowledge to conditions of factory work.

When the promised exchange of officials in the Home and Oversea State Departments takes place, it may be of considerable value to the future of the workers in factories in Australia to have a visit from a medical officer who has specialised in industrial hygiene.

Although England has maintained her reputation for giving a lead to reform in industrial hygiene, as in other branches of sanitation, yet Germany and the United States have both devoted considerable attention to the subject.

Both these countries have now established permanent expositions or exhibitions devoted to social and industrial betterment of wage-earners. At the Berlin exposition every safety appliance which inventive genius has devised can be seen in operation. According to the report of the United States of America's President's Homes Commission, the different labour unions appear to profit immensely by the special lectures and demonstrations which are given on Sundays, or, upon request, at any convenient time, by men formerly employed in " dangerous occupations."

Apart from safety devices for machinery and appliances for removal of dust and injurious gases, all improved methods calculated to diminish danger, as, for example, in the manufacture of white lead, &c., are illustrated by models and descriptive text, printed leaflets being distributed free of charge. Models, plans, and photographs of model-houses for workers, exterior and interior decorations, literature and charts concerning industrial betterment, all find a prominent place in the exhibit, which might with advantage be reproduced as a travelling exhibition, and be opened in every large industrial centre in both Australia and New Zealand.

This is another of the many branches opening out to State or Preventive Medicine, which, originally limited to the subject of hygiene, has called to its aid many branches of applied science, and has now become almost octopic in its range, touching on subjects of sociology, and even including economics within its grasp.

The trend of State medicine is towards the correlation and co-ordination of the various agencies which deal with the improvement of the environment and physique of the people.

Medical treatment of school children, including dental treatment, the natural corollary of medical inspection, is generally being introduced in various centres, thus bringing the work into line with that which has had such excellent results in Germany.

To-day we have Early Notification of Births Acts, women health visitors, together with school nurses and the employment of trained hospital nurses in visiting those getting charitable aid. Already the cry is to take hygiene to the home, and one sees on all sides a great awakening of the sanitary conscience of the people.

The establishment in different countries of infant milk depôts, schools for mothers, and various other agencies for lowering the infant death-rate have already had a marked effect in English-speaking and other countries.

In England, the passing of the Town Planning Bill has abolished for ever the insanitary back-to-back house, and will gradually lead to the improvement of urban areas. It behoves those responsible for the public health in our newer countries not to lag behind in this general advance.

The Majority and Minority Reports of the Royal Commission on the Poor Law in England have drawn attention to the urgent need for reform in the provision of medical attendance and treatment on a more rational system than that which has hitherto been the practice.

The promise of State insurance, combined with the recommendations of the strenuous advocates for the combination of the Public Health Service and the Poor Law Medical Service in the Old Country, together with what has been called the abuse of the voluntary supported hospitals and the exploitation of the medical profession, has brought the subject of the provision of medical attendance as affecting the public health to the forefront of public discussion.

We have seen a united medical profession at Home assisting in solving this problem. The Medical Secretary of the British Medical Association, at the recent London Conference, after referring to the existing system of medical attendance by private practice, contract practice, charitable services and public services, stated that the conclusion was that all would be agreed that such were inadequate, and some other provision was necessary.

It was pointed out that one of the most important points was that medical treatment should be prompt. It was found that the elements that were found in practice to lead to delay in obtaining medical treatment were: First, the fear of the honest patient that he might not be able to pay the cost of treatment; and secondly, the ordinary inertia of human nature.

All would be agreed of the utility of the reorganisation of sickness and invalidity insurance. Whether the solution lay in a State, municipal, and medical service, as suggested by the Minority Report, or by the formation in each centre of funds to which the beneficiaries would contribute, and from which doctors should be paid for their services, the opinion was that the contributing patient should have the same freedom of choice of his doctor which he enjoys in private practice. It was suggested, as is obvious to all observers, that at present there was much disease which was preventable; that the existence and effects of preventable disease were a matter of legitimate concern, not only to the individual affected, but to the community generally; and that one of the practical means of prevention was to make medical services readily available.

Although conditions in Australia and New Zealand are vastly different than those in England, yet, although neither a prophet nor yet the son of a phophet, I foresee in Australia and New Zealand—more especially in the cities—the evolution of a scheme on similar lines to that which is being formulated in England. Although all schemes and systems have their limitations, yet my little experience of the unsatisfactory nature of lodge work, both to the patient and to the doctor, makes me inclined to think that until some more rational system of medical attendance is evolved, if such be possible, State insurance with medical attendance subsidised by the central government, is the best solution of the problem for all concerned.

Whilst conscious that I have only skated round the outside edge of so wide a subject as Conditions of Work and Health, and sometimes have ventured to tread on thin ice, yet I know that in the hinterland of your minds ideas for reorganisation and improvement, with a view to more co-ordination and uniformity in health administration, are developing, and that time will probably show not only the truth of the aphorism of Aristotle, that " Physical health is the basis of mental health," but also that it is clearly related to national wealth.

THE HOUSE-FLY (*Musca domestica*) AND ITS IMPORTANCE IN THE SPREAD OF TYPHOID FEVER.

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Most public health workers believe that the ordinary house-fly plays at least some part in the transmission of typhoid fever. The debatable point appears to be its importance in this connection.

I. Some distinguishing, anatomical, and physiological features.

The normal length of the house-fly is about a quarter of an inch (6 to 7 mm.). It is easily distinguishable by the following:—In the dorsal side of the thorax there are four sharply defined (in front) black stripes on a dusty grey ground, which has a golden shimmer; its abdomen is buff yellow, with a dark median dorsal stripe, and the sides of the posterior segments are overspread with a reflecting grey; space between eyes in male scarcely one-fifth, in female nearly one-third of total width of head; proboscis is not visible from above; in the wing the fourth longitudinal vein is bent sharply upwards so as to nearly meet the vein above.

At anterior end of head there is a proboscis through which food is sucked up. The œsophagus passes from proboscis through the cephalic ganglion and neck into the thorax. At the junction of the anterior and middle thirds of the thorax it divides. One part, the crop duct, is continued backwards into the anterior part of the abdomen and becomes expanded into the crop, and the other passes into the proventriculus, which is situated immediately above the bifurcation. The crop is a bilobed sac, capable of considerable distension, which when greatly distended loses its bilobed shape and occupies a large portion of the the antero-ventral region of the abdomen. Its walls exhibit unstriped muscular fibres. The proventriculus, into which one branch of the œsophagus passes, is a curious, circular organ, flattened dorso-ventrally. Beyond the proventriculus is the ventriculus or chyle stomach, followed by the intestine and rectum. These points are easily made out on the diagrams I am exhibiting. (*See* Plate II.)

The crop and proventriculus are of considerable interest in connection with the distribution of infectious material by flies. At the commencement of a meal the crop is first distended with liquid food. If the feeding be continued, after the crop is fully distended, the food may pass directly into the ventriculus through the proventriculus. If, on the other hand, the fly is disturbed before any portion of the food has entered the intestine, the food which has been sucked into the crop is gradually passed into the ventriculus. In any case, after a variable period of time, the contents of the crop passes into the intestine. The proventriculus seems capable, therefore, of being closed during the early part of the meal in order that the food may not enter the intestine but pass into the crop. On complete distension of the crop, it opens in order to allow food to pass directly from the proboscis to the intestine. It also opens when it is necessary to allow material to pass from the crop into the intestine. After a meal flies usually regurgitate some of the fluid contents of their crops through the proboscis, and, no doubt, during this process the lumen of the proventriculus is closed in order to prevent the fluid from passing to the intestine. The proventriculus is variously regarded as merely a gizzard (Lowne), as having a pumping function, and also acting as a valve (Gordon Hewitt), as having a valvular function (Giles), as a valve possibly controlled, at will, by the fly (Graham-Smith).

Capacity of crop varies between 0·003 and 0·002 c.c.

About 90 per cent. to 98 per cent. of the flies usually found in dwellings belong to the species *Musca domestica*.

II. Life-history of the House-Fly.

The house-fly undergoes a complete metamorphosis, in which there are four well-marked stages, viz. :—

(i) The egg (ovum), commonly known as "flyblows."

(ii) The larva or maggot stage.

(iii) The pupa or chrysalis stage.

(iv) The imago, or perfect fly (the final stage). (*See* Plate I.)

1st stage.—The eggs are laid in small irregular clusters or in large collective masses. They are almost invariably deposited on or in such substances as will provide food for the larvæ or maggots, and preferably in fermenting vegetable matter or the refuse lying immediately over such materials, or in refuse that is likely to ferment.

The egg is chalky-white, and presents a highly polished surface due to the clear viscous substance with which it is coated. It is elongate, cylindrical oval, rather more pointed at front end, about one-twentyfifth to one-twentieth inch in length.

The number of eggs laid by a single fly at a time averages from 120 to 140. More than one batch (probably up to six) may be laid during the life of the fly.

2nd stage.—The larva is a white, footless maggot, measuring, when full grown, nearly half an inch in length. It can be distinguished from similar larvæ by examining under a microscope or powerful lens the posterior (broader) end of the body, when the openings there situated, through which the larva breathes, will be seen to be in the form of two pairs of very sinuous clefts, each pair of clefts being surrounded by a thickened plate with a straight inner and a semicircular outer edge.

The larva moults twice, thus dividing its existence into three instars or stages. During the third instar the larva grows rapidly.

3rd stage.—The pupa or chrysalis is at first of a pale yellow colour, but rapidly changes to a bright red and finally a dark chestnut colour. It is barrel shaped, regularly cylindrical, but tapering somewhat in front; one-fifth to rather more than one-fourth inch in length.

4th stage.—The perfect fly escapes from the pupa by breaking away the anterior end; this it accomplishes by inflating the frontal sac, which is situate in the front portion of the head between the eyes. By the inflation of this sac the fly is also enabled to work its way through the manure, ashes, &c., into the open air. When once it has liberated itself the wings develop, and when the integument has sufficiently hardened, the fly takes to wing. Flies do not grow; the size depends upon that of the larva when about to pupate.

Rate of Development.—The shortest period from the laying of the egg to the development of the fly has been given as eight to nine days (Griffith and Gordon Hewitt). According to most authorities a more common period is from twelve to twenty days. Ernest E. Austen, of the British Museum, in Local Government Board Report, New Series, No. 5, 1909, gives the periods of the various stages of the house-fly developed under favourable conditions, as follows:—

Egg.—Hatches in from 8 to 24 hours.
Larva.—4 or 5 days.
Pupa.—3 to 5 days.

Rate of development is influenced by a number of factors, such as temperature, moisture, character of food, fermentation:—

(1) Temperature is the most important. The most rapid development appears to take place at a temperature of about 32° to 35° C.; but according to the degree of cold the development from egg to perfect insect may take as long as forty-eight days or more.

(2) Moisture hastens development, as the larval stage is completed much more quickly in the presence of moisture. At the same temperature, larvæ kept on moist horse manure took only thirteen days to develop into flies, whereas those kept on dry horse manure took thirty days.

The pupæ do not require moisture, in fact the larvæ usually seek a dry position, such as the edge of manure heap, &c., when about to change into pupæ.

Sudden drops of temperature are very fatal to fly larvæ.

(3) Character of the food also affects rate of development. Larvæ develop more quickly on some substances such as horse manure than they do on others.

(4) Fermentation.—The presence of fermentation is very important for rapid development.

In case of eggs deposited on unfavourable media, not only may the developmental stages be delayed, but the resulting flies may be dwarfed.

The rate of development being a matter of considerable importance, I experimented with a number of batches of the Sydney house-flies during last summer. For this purpose ordinary hurricane lamp globes were made use of. The smaller end of the globe was covered with fine-mesh gauze, and the larger end was placed in wet sterile sand, in a saucer. About six to twelve flies were placed in each globe, food was placed inside the globe on the wet sand, a label for writing any phenomena observed was gummed on to outside of globe, and the whole was placed in the incubator with door open, and kept at a fairly uniform temperature. As food I tried horse dung, beef, mutton, and bread, but it was only with bread that I had any success. The horse dung soon got mouldy, while on beef and mutton the larvæ soon died. In my first trial a large number of blow-flies' larvæ hatched out, although there were only *Musca domestica* in the globe. This was due probably to a blow-fly having lighted on the gauze covering the glass and dropped its eggs through the meshes. To prevent a repetition of this kind of contamination in subsequent experiments I placed a square piece of cardboard over centre of gauze at top of globe, and inverted a glass funnel over the whole, and covered the upturned end of funnel with fine gauze. This arrangement, while not cutting off the air supply, prevented blow-flies dropping eggs into the globes.

I will only give one set of observations, namely, that showing the most rapid rate of development obtained in my experiments:—

Experiment 1.—

Ordinary house-flies were placed in captivity and incubated at a temperature of 28°–30° C.	18 Nov.,	1910
Eggs found	21 ,,	1910
Larvæ found	23 ,,	1910
Pupæ found	29 ,,	1910
Flies hatched	3 Dec.,	1910

Experiment 2.—

Flies hatched in experiment 1 placed in a fresh globe and incubated at a temperature of 30° 34° C. Apparently started pairing at once (2 days after hatched)		5 Dec.,	1910
Eggs found	4 p.m.	9 ,,	1910
Larvæ found	Noon	10 ,,	1910
Pupæ found	9.30 a.m.	15 ,,	1910
Hatched flies found	9.30 a.m.	19 ,,	1910

Thus from deposition of egg to hatching of fly took something under ten days.

It is interesting to note here that flies hatched on the 3rd December, 1910, appeared to be pairing, and, therefore, sexually mature on the 5th December, 1910—*i.e.*, in two days.

These observations were made under artificial conditions. It is possible that under natural conditions, in fermenting horse dung, &c., the rate of development is considerably reduced.

The possible duration of the *life of a fly* is difficult to ascertain. Dr. A. Griffith, of Hove, kept a male fly alive in captivity for sixteen weeks.

The chief breeding-places are collections of horse manure, spent hops, bread, decayed fruits and vegetables, excreta of all animals (including man) into whose dietary vegetable food enters, in rotten flock-beds, straw-mattresses, old cotton garments, rotten socks, waste paper, &c. Large accumulations of fresh horse manure are the most favoured breeding-places. It has been estimated (L. O. Howard, U.S. Department of Agriculture) that 1,200 or more house-flies will issue from a pound of horse manure. Municipal tips are important breeding-places. Human fæces are also favourite substances for breeding in. Aldridge hatched out 4,000 flies from one-sixth ($\frac{1}{6}$th) cubic foot of human fæces. Smith produced them with ease from the soil beneath all sorts of excrement. 500 flies have been bred from one evacuation of the human bowel.

III. Food and Habits of the Perfect Insect.

The feeding-places of flies are not necessarily their breeding-places. They feed on manure, sputum, most varieties of human foodstuffs, &c., but sweet substances, fresh manure, and human fæces are their favourite foods. The almost persistent habit flies have of feeding or alighting upon human excreta and then visiting the food of human beings, of course, constitutes their danger as transmitters of disease. Moisture is essential, and without it flies soon die. I have found flies die in captivity very quickly when the supply of moisture (water) has failed.

When undisturbed, flies gorge themselves in half a minute or less.

Under ordinary conditions fluid (food) begins to pass into ventriculus within ten minutes, and may be found throughout the intestine in two or three hours. At high temperatures it passes more rapidly. The crop, however, is not completely emptied for many hours. If flies are allowed constant access to food, material (coloured) from the first meal taken while under observation will be found to remain in the crop for many days.

After gorging themselves, flies usually move from place to place, frequently stopping to rub one leg against another, or to clean their heads and wings by passing their legs over them. At intervals, however, they sit still, and if they have fed on fluid materials, large drops of fluid exude from the tips of their proboscides. These drops gradually enlarge, until they are about equal in size to the insect's head. After a longer or shorter period the drop is slowly withdrawn or deposited on the surface on which the fly is standing. When deposited the drops gradually dry, and each gives rise to a round stain with an opaque centre, surrounded by a clearer zone, bounded by a distinct thin more opaque marginal ring. Flies often suck up the drops deposited by other flies. When flies feed on soluble solids, such as sugar, fluid is first deposited on the solids and then strong sucking movements take place. Dr. Graham-Smith's experiments seem to prove that this deposited fluid is liquid regurgitated from the crop unless that organ is empty, in which case the fluid consists of saliva alone. If infected food has previously been imbibed by the flies, the regurgitated fluid usually contains the infecting organisms.

Flies which have had access to abundant food *defæcate frequently*. The fæces, consisting of thick brownish or yellowish semi-fluid material, are deposited in single masses and quickly dry, forming opaque, raised, rounded stains. Occasionally the stains are pear-shaped. The fæces of flies fed on infected material have been found to contain the infecting organisms for many days after the feeding.

The number of deposits of regurgitated fluid and fæces of well-fed flies is extraordinary. This is shown by a series of experiments carried out by Dr. Graham-Smith. He states—"In the first series (A) ten flies were given a single feed of milk. When all had fed they were transferred to a fresh cage. At intervals the flies were again transferred to other fresh cages and the deposits in the old cages counted. In the second series (B) the deposits of eleven flies were counted in the same way, but milk was always in the cage, so that the flies could feed as often as they wished. The following table illustrates the number of deposits left by the flies :—

Time after feeding.	Series A.			Series B.		
	Regurgitated fluid.	Fæces.	Total.	Regurgitated fluid.	Fæces.	Total.
1st hour	30	11	41	22	10	32
2nd and 3rd hour ...	13	3	16	31	9	40
4th hour	18	6	24	6	4	10
5th hour	15	9	24	12	6	18
6th–22nd hour ...	49	10	59	108	16	124
	125	39	164	179	45	224

" Each fly in series (A) produced an average of 16·4 ' spots,' in series (B) of 20·4. In another experiment, ten flies, which had been given one feed of milk, produced in nine hours 209 deposits (191 regurgitated fluid and 18 fæces), and in the complete twenty-four hours 307 deposits (282 regurgitated fluid and 25 fæces), or an average of 30·7 'spots' per fly.

" No doubt the rate at which flies produce deposits depends on several factors, such as temperature and the form of food, &c. Flies are more lively in hot weather, or when placed in an incubator. That the kind of food exerts a considerable influence is shown by the following experiment. Three lots of flies were fed on syrup, milk, and sputum respectively, for several days. Those fed on syrup produced an average of 4·7 deposits per fly per day, those fed on milk 8·3, and those fed on sputum 27·0. In the latter case the fæces were much more voluminous and liquid than usual, and in fact the flies seemed to suffer from diarrhœa."

It will be shown later on that in the case of flies fed on infected food observers have found the infecting organisms in great numbers in these fly deposits (fæces and regurgitated fluid). Their importance cannot, therefore, be overestimated.

IV. Range of Flight.

The distance a fly can travel is of great importance. Dr. Miles Arnold found that flies could travel a distance of 190 yards.

Dr. J. T. C. Nash found that from large breeding-places, such as tons of refuse, flies will travel in countless battalions to the nearest houses which may be 200 or 300 yards distant. He found that in warm weather they travel further, and they will be found in considerable but decreasing numbers in houses within one-third to half a mile distant. Each street and terrace of houses formed a place of arrestment, provided there is abundance of pabulum, whether in the nature of filth or ordinary articles of human diet. Where few or no houses intervene, flies in large numbers will travel considerable distances—even over half a mile.

Dr. Copeman, Mr. Howlett, and Mr. Merriman, of the Local Government Board, England, observed a range of flight of marked flies, up to nearly a mile (1,700 yards) in the direction of the prevailing wind. They obtained two marked flies at a distance of 800 yards from the point at which they were liberated—one thirty-five minutes and the other forty-five minutes after being liberated.

Wind affects the range of flight as flies will, of course, travel further with the wind than against it.

V. The House-Fly in Winter.

Cold is fatal to flies. As the winter approaches, flies become sluggish and then disappear, or almost disappear from houses. How do flies pass the winter, in the fly or the pupa state? This question does not appear to have been satisfactorily settled. The majority of authorities think the cold season is bridged over by living flies and not by pupæ. Dr. Monckton Copeman states "the evidence at present available on this point appears to indicate that, in addition to the more or less active individuals which are usually to be found throughout the winter months in bakehouses and other warm situations, considerable numbers of flies in the adult stage hibernate outside dwellings in various sheltered situations." But according to Griffiths, flies will breed in winter under suitable conditions.

VI. Evidence incriminating the House-Fly as a disseminator of Typhoid Fever.

(*a*) *Epidemiological evidence.*—According to medical literature flies have been suspected of being active agents in the transmission of typhoid bacilli for many years, but they appear to have first received prominent notice in this connection at the time of the Spanish-American War.

The American Army Commission on the prevalence of typhoid fever among soldiers during the Spanish War of 1898 considered there was no doubt that flies served as carriers of infection. Dr. Vaughan, a member of the commission, gives the following reasons for believing that flies were active in the dissemination of typhoid fever:—

(*a*) Flies swarmed over fæcal matter in the pits, and then visited and fed upon the food prepared for the soldiers in their mess tents.

(*b*) Officers whose mess tents were protected by screens suffered proportionately less than those whose tents were not so protected.

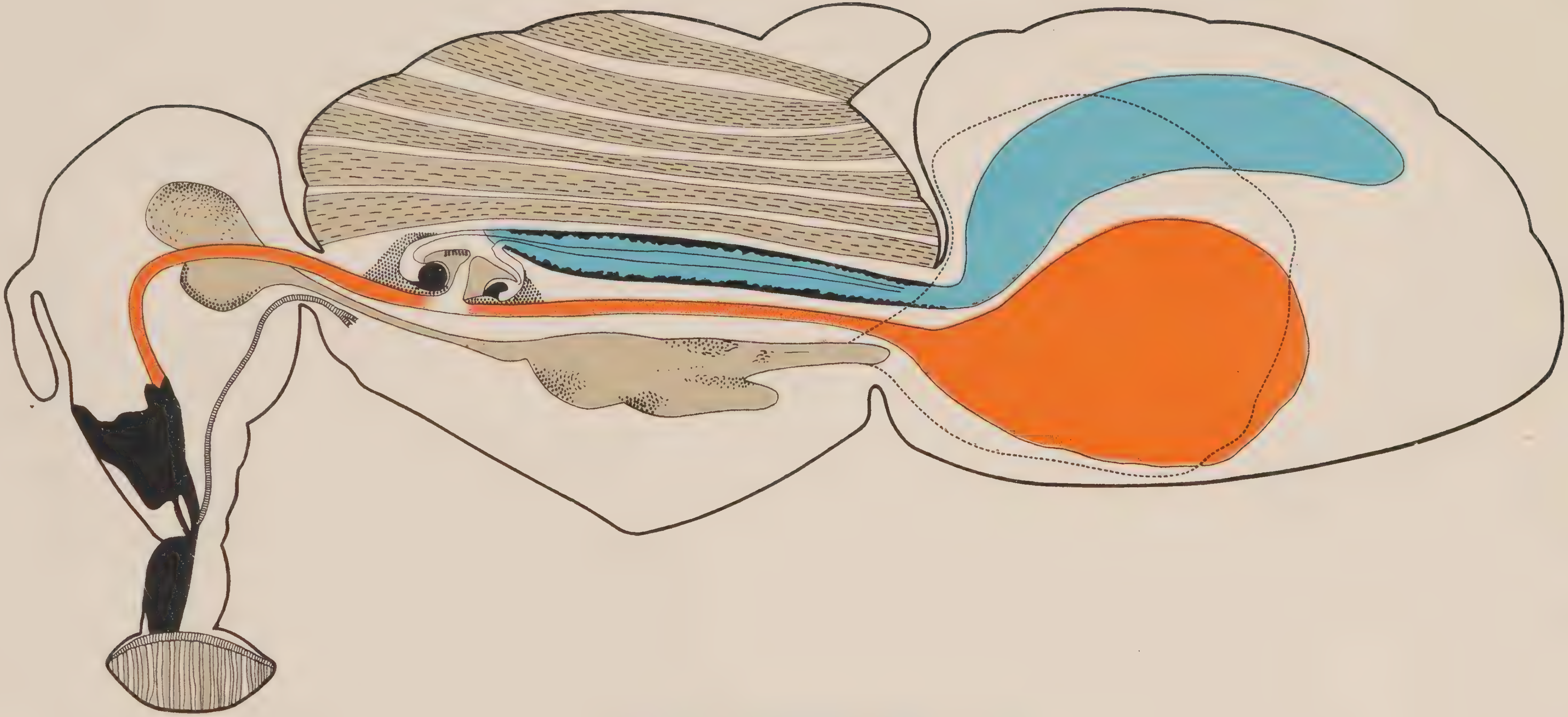
(*c*) Typhoid fever gradually disappeared in the fall of 1898 with the approach of cold weather, and the consequent disabling of the fly.

In connection with reason (*a*) the following statement by Major Edie might be mentioned:—"The cook at the division hospital (1st Division of the 3rd Army Corps) called my attention to the fact that when lime was being freely used around the pits the flies alighting on the food had their feet covered with lime."

The Commission considered flies had inflicted greater loss upon the American soldiers than the arms of Spain had.

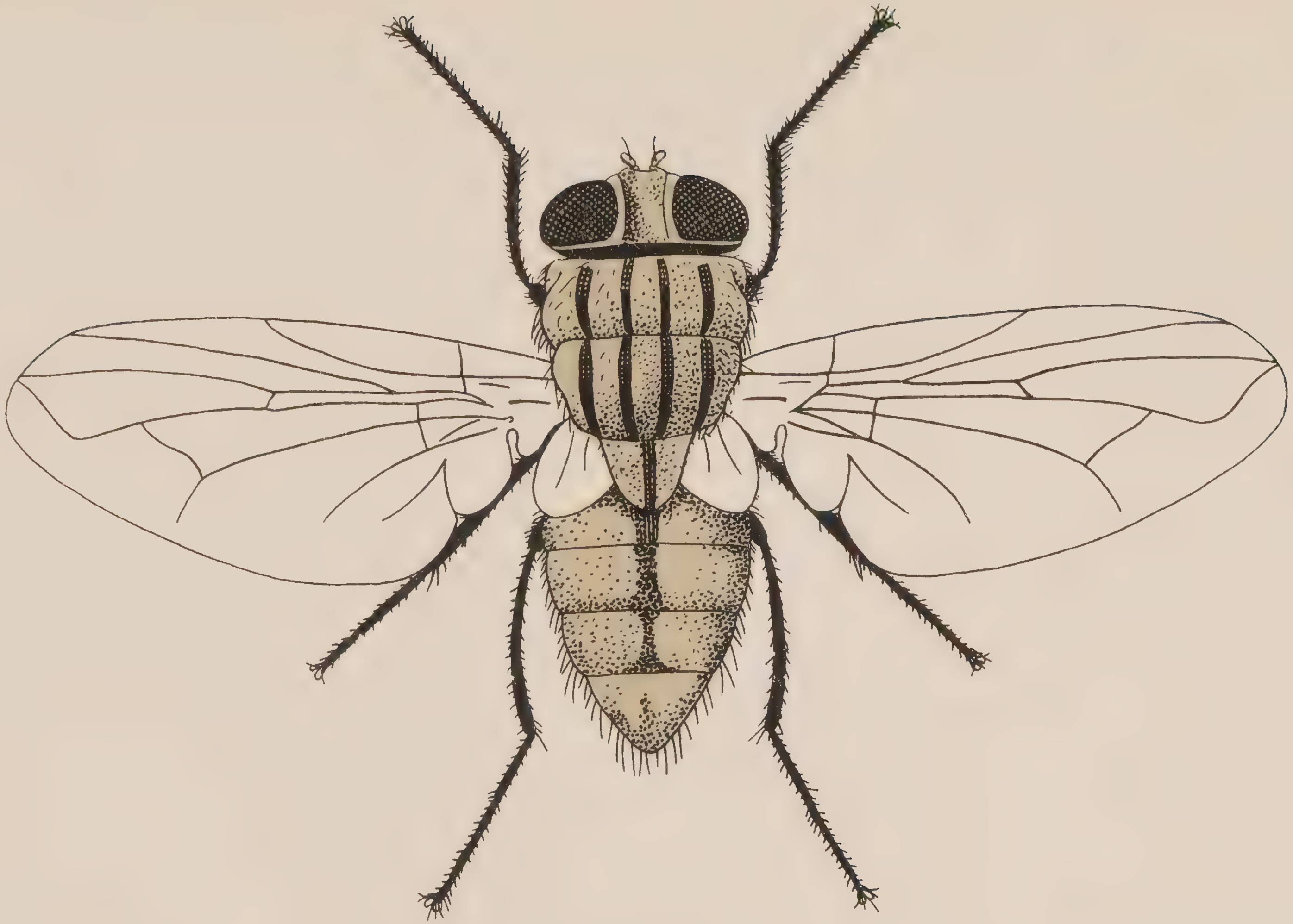
Captain Ainsworth, R.A.M.C., has shown how closely the seasonal incidence of typhoid fever and the prevalence of flies correspond in Poona. The same authority states that "many medical officers (in India) have made general observations to the effect that fly prevalence means probably an outbreak of enteric fever in the near future."

Dr. Niven, of Manchester, showed by careful and extended observations that in all probability flies were important factors in the spread of typhoid. He gives charts showing that after other causes, such as shellfish, &c., have been allowed for there is in Manchester a rise in the number of cases of typhoid corresponding with the increase in the number of flies caught.



Schematic Longitudinal Section of a Fly.

Taken from Reports to the Local Govt. Board on Public Health and Medical Subjects, N.S. No. 40.



The Boer War also provides testimony in favour of the very distinct relation of cause and effect between the fly and typhoid fever. Dr. Arnold H. Watkins, writing from Kimberley, stated—" Flies swarm on all fæces exposed to the air, and they also swarm in all milk and on every article of food left for a minute uncovered. It is a very simple process for the fly passing from the typhoid stool to the milk, the jam, the butter, or other articles of food, to carry enough fæcal matter to infect the consumer of the food. With the advent of cold weather the flies completely disappear, so does, almost entirely, the typhoid."

Dr. A. B. Dunne wrote—" The plague of flies which was present during the epidemic of enteric at Bloemfontein in 1900 left a deep impression on my mind. Nothing was more noticeable than the fall in the admissions from enteric fever coincident with the killing off of the flies on the advent of the cold nights in May and June."

Tooth and Calverley give evidence to a like effect.

Much evidence comes from India. Lieutenant-Colonel Caton Jones, R.A.M.C., pointed out in 1907 that since a raid had been commenced in Nasirabad in 1904, the enteric rate there had very much diminished.

Major N. Faichnie, R.A.M.C., reports that this improvement in the enteric rate at Nasirabad had been maintained up to the time of writing (August, 1909). He also reports that there was a sudden and permanent diminution of enteric fever at Mhow following on steps being taken to prevent flies breeding at the night-soil trenching grounds. This authority also states—" At the beginning of this year (1909) the only station in the division that was suffering from enteric fever was Jubbulpore, which has an unquestionable water supply, but which is swarming with flies, even in cold weather."

Medical literature contains a large amount of evidence of this nature.

The marked diminution of typhoid in cold weather, corresponding to the disappearance of flies, is an important point. Cold does not kill the *B. typhosus* and carriers continue excreting the bacilli in winter, as instanced by the case reported by me last year (*Australian Medical Gazette*, August, 1910) as being effective in July, but there are very few flies to carry the infection.

Dr. J. S. Purdy reported an epidemic of typhoid fever occurring in Auckland, New Zealand, in 1908, as due to flies conveying infection from the discharges of a typhoid patient which had been left exposed in a wooden receptacle, owing to neglect of the scavenger.

Early in 1910, I, acting under instruction from the Chief Medical Officer of the Government, investigated an outbreak of typhoid fever at Eugowra, a town about 250 miles west of Sydney. Population of the town, 500; living in eighty houses. Each house was provided with a pan closet, the pan being taken away once a week, and replaced by an empty one. No steps were at that time taken to keep the night-soil in the pans covered. Onset of illness in the first case was notified as 3rd January, 1910, and that of the last case as 14th March, 1910. During the outbreak, which thus lasted about ten weeks, there were, according to notifications received, seventeen cases in the town and two cases a short distance outside.

Both milk and water were easily excluded as casual agents, because most of the cases either obtained milk from their own cows or used condensed milk, and all obtained their water from tanks or wells on their own premises. Contact infection did not play a very great part in the outbreak. The conveyance of infection by flies from uncovered infected night-soil to food appeared to be the most important, if not the only cause of the epidemic.

On my arrival at Eugowra, which happened to be at lunch time, I was struck with what can only be described as a plague of flies. The flies were so bad that at the hotel where I stayed two saucers were usually brought in with each cup of tea, one to place over the top of the cup to keep the flies out, also some of the boarders at meal times placed their bread in their coat pockets and ate it from there to keep it from the flies. On making inquiries around the town I learnt that no such plague of flies had previously been experienced by any of the inhabitants. It was necessary then to seek for its cause. It was found that during December, 1909, and the early part of January, 1910, the scavenger had indulged in a drinking bout, lasting some weeks, during which time pans were not removed, and in many cases overflowed; also a number of full pans were allowed to remain uncovered in the scavenger's yard in the centre of the town. Thus was provided, at the most suitable time of the year, excellent breeding material for flies.

Naturally the next thing to do was to find out where the original infected material came from. After some difficulty I found that A.D., the first case of the epidemic, returned to Eugowra from a trip to Sydney about the middle of December, 1909, infected with typhoid fever. The date of onset of illness as shown in the notification certificate would appear to negative the conclusion that this man was infected on his return from Sydney, but I was able to ascertain that he was definitely ill, though not confined to his bed, for fully a fortnight in December. I was also able to get reliable evidence that this man, during the period of his illness previous to taking to his bed, was in the habit of using the closets at two hotels, a billiard saloon, and at relatives' houses. This period, it will be observed, corresponded to the time when the pans had been allowed to remain for weeks unemptied.

I visited a large proportion of the houses in the town, and nowhere did I find any very serious attempt made to keep food from fly contamination. We thus had all the conditions favourable for a fly-borne epidemic, viz., quantities of infected fæcal matter in different parts of the town for the flies to feed on and breed in, and human foodstuffs in the houses easily accessible to the infected flies.

The following additional evidence was obtained in favour of the conclusion that this epidemic was caused by flies carrying infected material from the privies. Just before my arrival at Eugowra the local authority appointed a new sanitary inspector (the office had been vacant for some months). This man, although quite unqualified, turned out to be very intelligent. I impressed upon him the danger of flies carrying infection and the necessity for all fæcal matter to be covered up as soon as it was deposited in the closet pans. He set to work and made every householder, not only provide sawdust in his privy (there happened to be a saw-mill in the town), but also saw that this sawdust was freely used in the pans. When I revisited the town the third week in February, 1910, I did not see a single particle of uncovered fæcal matter in any closet, although I visited many for this purpose. No other methods of immediate practical value were adopted to combat the epidemic, but it ceased in about three weeks after the people had been educated up to keeping the contents of their closet pans covered.

Charts have been prepared by various observers (notably Captain Ainsworth and Dr. Niven) to illustrate by means of curves the relationship between typhoid incidence and the prevalence of flies.

Ainsworth, in India, prepared charts in which the curves are very striking, the fly curve reaching its acme about two weeks before the maximum number of cases of typhoid occurred.

During the last typhoid season (1st September, 1910, to 31st May, 1911) I had fly enumerations made in Sydney and in several country towns. Charts herewith, comparing the fly prevalence thus obtained with the typhoid incidence, proved to be of no value. This was to be expected, as the numbers dealt with were too small.

(*b*) *Bacteriological evidence.*—An enteric stool, according to Firth and Horrocks, may contain from three to five typhoid bacilli in 0·006 milligrammes. When we consider the number and volume of evacuations from a single patient, the fact that typhoid bacilli may be present in the stools during the incubation period, and that a considerable number of persons continue to excrete typhoid bacilli for long periods after an attack of typhoid, we must be struck with the enormous amount of infected material there is at least in unsewered districts, from which flies could obtain typhoid bacilli. This amount is further increased by the excreta of undiagnosed cases of typhoid. That these undiagnosed cases form a large proportion of the total number of cases was shown by Dr. Niven, who found in Manchester, in 1905, that about one-sixth of the cases were overlooked.

Numerous observers have shown that typhoid and other bacilli can be recovered from the bodies of flies allowed to walk over infected material, such as cultures of the various bacilli. Seeing that flies are covered with numerous minute hairs they must become contaminated in this way. This method of carrying the bacilli is, however, probably of least importance, as the bacilli on the flies' bodies would soon be killed by exposure to sunlight and wind.

Ficker, in 1903, recorded the fact that he isolated *B. typhosus* from flies caught in a house in Leipzig where eight cases of typhoid had occurred.

Dr. Alice Hamilton, in connection with an epidemic of typhoid fever in Chicago in 1902, inoculated eighteen tubes with flies caught in two undrained privies, on the fences of two yards, on the walls of two houses, and in the room of a typhoid patient, and obtained typhoid bacilli from five of these tubes.

Ficker recovered *B. typhosus* from flies twenty-three days after they had been infected.

Dr. Graham-Smith, after feeding flies on syrup infected with *B. typhosus*, obtained the bacilli from the intestinal contents of these flies up to six days. He also obtained the bacilli from the fæcal deposits of the flies up to forty-eight hours. Further, forty-eight hours after feeding on infected syrup, flies infected plates over which they walked.

As the typhoid bacilli do not survive more than a few hours (5–18) on the legs and wings of flies, it was concluded that the infection in this case was due to the fact that flies frequently attempt to suck the surface on which they happen to be, and in so doing infect it with fluid regurgitated from the crop. Dealing with *B. prodigiosus*, he found that clean flies may infect themselves by feeding on the deposits left by infected flies.

Dr. Graham-Smith states—"It should be remembered that a fly may cause relatively gross infection of any food on which it alights after having fed upon infective substances, be they typhoid, cholera, or diarrhœa stools. Not only is its exterior contaminated but its intestine is charged with infective material in concentrated form which may be discharged undigested upon fresh food, which it seeks. Consequently the excrement voided by a single fly may contain a greater quantity of infective agents than, for instance, a sample of infected water. In potential possibilities the droppings of one fly may, in certain circumstances, weigh in the balance as against buckets of water or of milk."

Major Faichnie, R.A.M.C., states—"I have found *B. typhosus* in flies from Sehore, once; from Kamptee, twice; from Nasirabad, once in flies from the bungalow of an officer who had enteric fever, and once from flies in the officers' mess there; from Nowgong, twice—once in flies from the Royal Artillery coffee-shop, and again in flies from the trenching ground, making a total of nine times in three months. Except those from Nasirabad, the flies were always flamed before examination, and a control of the washed flies was taken before crushing, so there is no doubt the bacillus was actually in the interior of the fly, most probably in the intestine."

This authority considers that neither the feet of flies, nor their excreta when they feed on the contents of latrines, are common vehicles of fly-borne infection; but, on the other hand, the chief and most common method is by excrement when flies are bred in an enteric-infected material. He explains that by this he means that one station may swarm with flies, bred only from the excreta of cows and horses, and yet have no enteric; while another place where there are very few flies, but where these are bred from human excreta, either in or out of the station, may have an epidemic, the source of infection being the excrement of the flies and the insects themselves being carriers. This, he states, "explains many conclusions formerly difficult to accept. In a word it is the breeding-ground that constitutes the danger, not the ground where the flies feed." He found, as the result of some highly interesting breeding experiments, that out of thirteen flies bred from a typhoid stool, at least six contained *B. typhosus* in their intestines; and the bacillus was recovered from the intestines and excrement of a fly sixteen days old. Plates prepared from larvæ bred in human fæces have the same appearances as those put up from the fæces the larvæ were bred in.

(*c*) *Combined Epidemiological and Bacteriological evidence.*—The Annual Report of the Sanitary Commissioner with the Government of India for 1909, page 50, contains the following statement:—"In the 94th Russell's Infantry one or more cases (of typhoid fever) occurred in every month except January, February, and October. An attempt was made by the sanitary officer of the Mhow Division to ascertain the vehicle of infection.

His report states that after excluding water, milk, and personal infection, it was decided that flies were the most likely cause, and when an examination of some caught in the lines was made in December, *B. typhosus* was found inside them."

In 1908, Dr. Heywood Wilshaw, England, investigated an outbreak of typhoid, and reported—"After the occurrence of a case of enteric fever in a house forming one of a row, a number of typhoid cases made their appearance in the neighbouring houses. All known channels of transmission, for example, personal contact, defective drainage, polluted water or milk, were excluded. The only condition common to all the houses of the row was this—that they were swarming with flies." Some of the flies were submitted to Dr. E. Klein for bacteriological examination. He found *B. typhosus* in the flies examined, and reporting on the matter states—"The actual demonstration of the *B. typhosus* in the flies lends, therefore, strong support to the conclusion arrived at from the epidemiological data—namely, that the contagium in the cases of enteric dealt with in Dr. Wilshaw's report was conveyed by the flies of the locality."

Major Faichnie in 1909 investigated a small outbreak of typhoid at Ramptee, where, after excluding all other causes he was obliged to suspect flies. These insects were by no means numerous. About forty were collected, twenty each from the verandahs of the artillery and infantry kitchens. Typhoid bacilli were found in the intestines of each of the two sets of flies. Cultures of these bacilli were sent to Lieutenant-Colonel Semple, Director of the Central Research Institute, Kasauli, who stated they were undoubtedly typhoid bacilli. Amongst other tests he immunised rabbits with these cultures and found their serum agglutinated stock cultures of *B. typhosus* in high dilutions.

VII. Precautionary Measures.

The measures to be taken to prevent transmission of typhoid fever by flies may be grouped under four headings, viz.:—

(1) Prevention of access of flies to infected material.
(2) Prevention of flies breeding.
(3) Destruction of adult flies.
(4) Prevention of food contamination by flies.

(1) **Prevention of access of flies to infected material.**—The most important infected materials are human fæces and urine. Where the pan system is in vogue some material, earth, sawdust, &c., should be provided in each privy, and each detail covered up immediately after pan is used, and the seat-lid kept closed except when privy is actually being used. The use of such substances as blue oil or kerosene is an important aid in keeping flies out of the pan. The pan may be swabbed out with one of these substances each time after it is emptied and cleaned, or a little may be poured on to the material used to cover up the details in the pans.

Night-soil, and especially that from infants, should not be allowed to lie about uncovered.

People frequently relieve themselves in the bush in the country, and it appears almost impossible to prevent them, but even in the suburbs one sometimes sees fæcal deposits on the less frequented footpaths.

In this connection attention must be called to the dangerous practice existing in our trains. A person, who may be either a "carrier" or in the incubation period of typhoid fever, uses one of the train W.C.'s, he pulls the string and the excrement is flushed on to the railway line to be immediately attacked by numerous flies. This may happen just as the train is passing through the centre of a town or village.

The vehicle used for removing pans should not be forgotten. Professor Stiles, of the United States Public Health and Marine Hospital Service, describes the important part these vehicles play as distributors of flies, and calls attention to the possibilities of thus distributing filth and infection. The insides of these vehicles should be frequently washed or sprayed with some substance such as crude carbolic, kerosene, &c., to keep flies off.

Patients suffering from typhoid quite possibly form foci for flies to obtain a supply of infecting material. Tooth and Calverley, referring to typhoid in camps during the South African War, state that—"In a tent full of men, all apparently equally ill, one may almost pick out the enteric cases by the masses of flies they attract. This was very noticeable at the Modder River, for at that time there were in many tents men with severe sunstroke who resembled in some ways enteric patients, and it was remarkable to see how the flies passed over them to hover round and settle on the enterics. The moment an enteric patient put out his tongue one or more flies would settle on it." Typhoid cases should be kept under mosquito curtains.

(2) **Prevention of flies breeding.**—To reduce the number of flies to a minimum, domestic and municipal cleanliness is absolutely necessary.

Stable manure should not be allowed to lie about in uncovered heaps, but kept in properly constructed, *covered*, dung bins. These bins should be thoroughly emptied and carefully swept at least once every seven days. Where it is necessary to allow manure to stand some time to "ripen" for garden purposes a layer of earth should be placed over the heap.

According to Newstead, the application of Paris green solution (2 oz. to a gallon of water) to stable manure will kill 99 per cent. of the larvæ, while an application of a 1 per cent. crude atoxyl in water kills 100 per cent. of these larvæ.

Kitchen refuse should be kept in covered, watertight receptacles. Such substances as bedding, straw, old rags, paper, dirty bedding from the "hutches" of pet animals, &c., should not be allowed to lie about, but be immediately burnt or otherwise disposed of by householders.

Councils should pay special care to the condition of rubbish tips. All inflammable material should be burnt each day, and the remainder of the refuse kept covered with a good layer of soil. A dry site should be chosen for a rubbish tip.

Accumulations of dust and dirt behind pictures, furniture, in corners, &c., should not be allowed in the home.

Fæcal matter should never be allowed to remain uncovered at night-soil depôt; special care should be taken to have the night-soil covered immediately after it is emptied into the trenches. Professor Stiles and Dr. Gardiner, of the United States Public Health and Hospital Marine Service, have shown that when flyblown fæcal material is buried under 72 inches of clean sand it is possible for adult flies to issue from the surface.

This raises a doubt as to whether the prevailing method of burying night-soil in shallow trenches with a covering of about 10 inches of earth is a sufficient safeguard.

Fowls (not ducks nor geese) are very fond of fly larvæ, and if allowed free access to fly-breeding places such as manure, garbage, &c., will help to reduce the number of flies by eating the larvæ.

(3) **Destruction of fully-developed flies.**—

(*a*) Fly traps.—There are several forms of efficient fly-traps on the market. Dr. Copeman recommends that traps of fine wire gauze, of rounded shape, generally known as "balloons" be used, side by side with fly-papers. The balloons he baits with a few grains of coarsest brown sugar, moistened with (but not dissolved in) a little stale beer. I have had good results with the use of fly-papers. The position in which these fly-papers are placed is of importance; to get the best results they must be placed in a well-lighted part of the room, preferably near the window.

(*b*) Chemicals—Formalin.—Weak solutions of formalin, 8 per cent. to 10 per cent., in water or milk and water, placed in an open vessel in a room, have been recommended, the idea being for the formalin not to act as a fumigant, but as a poison. The flies drink the solution and then die.

Mortein.—While making some inquiries at butchers' shops some time ago, I was informed that mortein frequently dusted about the shops, especially near the enclosed fronts caused a marked diminution in the number of flies. Other makes of insecticide would probably act as well.

There are some proprietary fly destroyers on the market, which act by supplying poison to the flies, *e.g.*, the Daisy Fly Destroyer.

Burning pyrethrum powder in a room will quickly clear the room of flies. The flies become stunned and fall to the ground, when they should be collected and destroyed.

(4) **Prevention of food contamination by flies.**—Household cleanliness reduces the number of flies in a house.

All foodstuffs should be kept covered. It is common enough to see the strictest precautions taken to keep milk covered, but we very frequently find that the solid and semi-solid food materials, such as sugar, cake, jam, &c., are allowed to remain freely accessible to flies. When we remember the physiology of fly feeding, and how fluid is regurgitated, often from the crop, to moisten solid particles before feeding on them, we can understand how important it is to keep these articles of food covered.

Wire screens to all openings in kitchen, pantry, and dining-room, especially those openings on the sunny sides, will keep out numbers of flies.

Newstead recommends the use of sun-blinds in all shops containing food which attracts flies.

VIII. Summary.

(1) House-flies are numerous in most houses in the warmer weather, and they have in the majority of cases free access to human food.

(2) Their anatomy favours the retention of live bacilli in their insides for days. That this retention does take place has been proved bacteriologically in the case of *B. typhosus* and other organisms.

(3) Their habits of frequenting and feeding on and breeding in human fæcal deposits places them in the possible position of being often loaded with typhoid bacilli.

(4) House-flies produce large numbers of deposits of regurgitated fluid and fæces every twenty-four hours. If the flies have fed on material containing typhoid bacilli, the typhoid bacilli may be contained in large numbers in these deposits for a number of days.

(5) The manner in which flies feed, especially when ingesting soluble solids, favours infection of human food with any bacteria the flies may have previously imbibed.

(6) There is a large amount of epidemiological, bacteriological, and combined epidemiological and bacteriological evidence pointing to the causal connection between flies and transmission of typhoid fever.

It would appear, therefore, that the house-fly (*Musca domestica*) is an important agent in transmitting typhoid at least in unsewered districts.

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MOSQUITO-BORNE DISEASE IN QUEENSLAND.

By E. Sandford Jackson, M.B., B.S. (Melb.).

In 1882 Dr. Joseph Bancroft drew my attention to some cases of Filaria sanguinis hominis. It was soon after my arrival at the Brisbane Hospital as Resident Medical Officer, and he also found some specimens of the filaria immitis of the dog, and demonstrated them to me and their embryos. Since that time, having been in constant practice inside and outside of the hospital, I have had the best opportunities of noticing the increase of the Filaria sanguinis hominis.

Eighteen years ago, impressed by the apparent increase of the disease, I wrote a paper on the "Manifestations of the Filaria sanguinis hominis in Queensland," and read it at the Queensland branch of the British Medical Association; and this was followed almost immediately by papers from the late Dr. Peter Bancroft and Dr. Thomas Bancroft. Interesting discussions arose at the reading of these papers, and I remember that at one of these Dr. T. Bancroft foreshadowed the connection between the mosquito and the human host, and the special manner of its transmission, which up to that time had been supposed to be through the medium of drinking water.

Then for many years there was no communication on the subject, except one from Dr. Flynn, of Ipswich, till three years ago. Then Dr. Maclean, of the Brisbane General Hospital, drew my attention to the increasing number of filarial conditions which were coming before us for operation, and urged that we should make a move. It had been found, in an examination of a few hundred cases, that a percentage of 17 per cent. of all the people admitted to the General Hospital were the possessors of microfilaria in their blood, and this quite independently of the particular complaint for which they were under treatment in hospital. Many of those showing the microfilaria in their blood had no evidence of any other kind that they were the subjects of filaria. Dr. Maclean and I then brought the matter before the Queensland branch of the British Medical Association; a discussion ensued, and a deputation approached the Health Commissioner of the day and was received with more or less sympathy. Since that time Dr. Maclean and others have done their best to keep the matter before the public. A further series of examinations of the blood of patients on their admission to the hospital and at various times about 2,000 have been examined by the resident medical officers, and the result has been that never less than 10 per cent. in the various batches have been found to have the microfilaria in their blood. The percentage has shown a slight decrease in the later batches owing to the fact that in those batches there have been a large number of immigrants—a larger number, in fact, than in the first batches examined. Of course the immigrant does not show the microfilaria in the blood.

So, then, the British Medical Association and its Queensland members have been since that time urging the destruction of mosquitoes systematically and thoroughly, and papers and discussions have been frequent. As time has gone on, and we have considered the matter more and more, it has become increasingly evident that the association of the mosquito and filaria is not the only reason for the destruction of the former. Of course, long before the crusade was begun by us it was evident that malaria was a sufficiently cogent reason for the destruction of the mosquito in the neighbourhood of settlement in North Queensland. Consideration and perusal of the latest literature made it abundantly evident that the connection of the mosquito with the disease dengue constituted another reason for the destruction of the former, and again, perhaps more than all, the fact that yellow fever was conveyed by the mosquito Stegomyia fasciata vel calopus made the destruction of the carrier of that disease a most important matter indeed, since on the opening of the Panama Canal it seems very unlikely that we shall escape having that disease introduced to Eastern Australia.

So far as Queensland is concerned, therefore we have known long enough the connection between the mosquito "Culex fatigans" and filaria, and also that between the anopheles mosquito and malaria there is enough of both these throughout the State of Queensland to warrant a move against the insect. Those of you who have never had any personal experience of life in a city where dengue is raging can, I think, have very little conception of the enormous upset to business and the untold misery of such an epidemic. Though in one epidemic a few years ago in the city of Brisbane and its suburbs there must have been not less than 100,000 cases in a few months, you have not heard very much about it from us, because, perhaps, the mortality is not very great, and because we are a hardy race, and have learned to put up with things that cannot be helped. It is certain now, however, that dengue is a thing that can be helped, and it has become to some of us a matter of the very greatest importance that we should be rid of it. The mortality was estimated at 1 in 1,000, but it must not be forgotten that there was no notification of the disease, and the estimate of the numbers was a mere matter of guess-work. In such fearful stress of work as we were put to in the epidemic I refer to it was difficult to get time to attend to one's meals, let alone take notes or keep records of any value.

The evidence connecting the Culex fatigans mosquito with dengue is complete enough to animate us with a desire that the insect should go. That this mosquito should bring us two such diseases, as it does, is more than we can bear.

The connection between yellow fever and the Stegomyia fasciata is, however, a much more important reason for a crusade against mosquitoes than any other. It is a reason that, so far as records of the presence of the Stegomyia fasciata goes, affects almost the whole seaboard of Eastern Australia. What applies to us also applies to the Pacific Islands and the southern parts of Eastern Asia. Throughout these parts that particular mosquito seems likely to be found. We can assure you that in Brisbane there is hardly a house which does not contain many individuals of this species. It is, I think, even commoner than the Culex fatigans in the suburban homes of Brisbane. The city itself is comparatively free from mosquitoes of all kinds, owing to the absence of water-tanks in the warehouses, &c. The stegomyia bites freely in the daytime, when people are not protected by mosquito-nets. Now, if the culex gave us an epidemic of 100,000 cases of dengue, it seems extremely likely that its little striped cousin (Stegomyia fasciata), if not interfered with, would succeed in producing an epidemic of a similar magnitude. There would be this difference, however: the mortality would be very much higher—not 1 in 1,000 only, but perhaps 500 in 1,000. It is not likely that we should have quite such a big epidemic, because we should wake up as soon as the first case occurred and start the crusade against the mosquito; but the mortality would probably run into thousands, as against a hundred or so in a big dengue epidemic.

With such considerations as the foregoing in view, it began to seem very necessary in the minds of many of the Brisbane practitioners that a very energetic attempt should be made to secure the earliest possible commencement of a campaign against the mosquito generally. It was certainly very necessary throughout coastal Queensland. It seemed also that the need affected the whole of the east of Australia and New Zealand, because it was believed that all these regions were more or less infested with the Stegomyia fasciata.

With this end in view, the agitation was kept up by the Queensland branch of the British Medical Association, and ended in June last in a public meeting organised by the branch, presided over by Sir William Macgregor, and addressed by public men belonging to the Association and others. Resolutions were passed affirming the desirability of a raid on mosquitoes, and were forwarded on to the Government by the Mayor of the city.

It is pleasant now to reflect that, as the result of the work of the Queensland branch of the British Medical Association, there will be introduced, this session of Parliament, a Bill to empower the Health Department to proceed; and as, in the opinion of the knowing, that Bill will also be passed, there is good prospect of the living conditions of Queensland being so improved at no distant date as to place us in the very front ranks of climate.

In a pamphlet which I circulated on the subject of "Mosquito-borne Diseases in Eastern Australia," I repeated the statement which had been made to me by various authorities, that the stegomyia was also to be found all over the east of Australia. That statement was at once challenged. I had sent my pamphlet to the President of the Congress, Dr. Pockley, and he at once wrote, telling me that the local entomologists had never observed them in Sydney. In effect, they stated that there were none of the Stegomyia fasciata vel calopus, though there were, in fairly abundant numbers, the allied mosquito, S. notoscripta. (This must be Scutomyia notoscripta.)

I at once put myself into communication with Dr. T. Bancroft, whom I have more than once to thank for very interesting letters, and with Mr. Tryon and Mr. W. R. College. All these gentlemen have done a great deal of work in matters entomological in this State. As a result of the correspondence with these gentlemen, I gather that none of them have seen the stegomyia in Sydney;

indeed, Dr. Bancroft tells me that he regards the absence of stegomyia as having some connection with the absence of dengue in Sydney, and its apparent inability to spread in that city.

Castellani and Chambers speak of it as being found all the world over, and elsewhere it is so described. It is proved beyond doubt that it is easily conveyed on board ship. It is beyond doubt that it is present all over Queensland and the islands of Torres Strait. It has, I believe, been reported from Victoria. If that report were correct, it would be strange that the insect does not exist in Sydney. Plainly, though, it would seem that it must be admitted that there are not many Stegomyia fasciata in Sydney; and it must always be remembered, as Dr. T. Bancroft reminded me, that it is easy for the authors of books on mosquitoes to be misled by the fact that collectors forwarding their specimens from a certain locality do not always label the specimen with the locality in which the specimen was found. The author naturally records the specimens as taken at the locality from which he received the letter accompanying them.

It is a matter, however, on which there should be no doubt—this question of how far south the Stegomyia fasciata is found. It is, moreover, of equal importance to determine whether the Scutomyia notoscripta has ever been the subject of experiment as a carrier of yellow fever in other countries, and with what success. For all the information we have here about him, he might prove as bad a carrier of yellow fever as the stegomyia.

I have said that the stegomyia is easily carried on board ship, and was originally, I believe, introduced to Queensland. So far as Brisbane is concerned, it is very certain that this variety has increased in numbers to an enormous extent in the last twenty-five years. In 1882, when I first came to Brisbane, the majority of mosquitoes in my house were night mosquitoes—the Culex fatigans probably. Now there are in most houses as many day-mosquitoes or more, and they are chiefly Stegomyia fasciata.

There are a number of allied questions which want clearing up in connection with mosquitoes and their ways.

There is, for instance, the puzzling one of why, if Sydney has many of the Culex fatigans, as she is said to have, the two diseases which seem to depend on it for carriage in Brisbane, viz., filaria and dengue, do not get a hold? Thousands of dengue cases must have found their way to Sydney from time to time, and yet there has been no extension of the disease in that city. Hundreds of filaria cases have gone south from Brisbane. I have never been able to hear of one case of either disease arising in New South Wales. I should be grateful if any of my hearers will correct the impression conveyed by that statement if they know of any case to the contrary. One of the health officers in New South Wales wrote me some years ago that he knew of some cases of filaria in that State, near Newcastle, but that in each case the individual had spent some time in Queensland. I suggested that in all probability other members of the families of these individuals had microfilaria.

It would, in my opinion, be very interesting to make an examination of the blood of a thousand consecutive admissions to a hospital in Newcastle and Sydney for microfilaria.

The following, then, are questions which require entomologists to answer:—

1. Why does neither dengue nor filaria become familiar in Sydney?
2. How far south is it possible to find these diseases arising endemically or epidemically?
3. What is the southern limit, if any, to the Stegomyia fasciata?
4. How far south is the Culex fatigans to be found? or any other of the mosquitoes which carry microfilaria?
5. Is the Stegomyia fasciata capable of carrying dengue? It would seem that the only reason why it is not more frequently a carrier of filaria is that it does not bite so much at night as in the day.

I have pointed out that the Stegomyia fasciata is easily conveyed by ships; that it was once much rarer in Brisbane than it is now; that, in fact, it was introduced to Queensland from elsewhere; that it exists in countless thousands in Queensland.

6. Is there anything to prevent the same mosquito being introduced into Sydney? Is there anything which prevents its spreading in Sydney?
7. Does the allied mosquito, Scutomyia notoscripta, which is common in Sydney as well as Queensland, exist to the east of America? and has any test ever been made of this insect as a carrier of yellow fever?

Now, it seems to me, and I am sure it does also to many of you, a matter of the gravest importance to the study of mosquito-borne diseases in the Australian States that some experienced entomologist should be told off and sufficiently subsidised to make a most exhaustive examination of these matters. Probably the work I refer to would be better begun at the Queensland end, and by someone familiar with the entomology of the subject in that State.

This work requires special knowledge, special experience, and special industry. The man who undertakes it will have lots to do for some years to come, and should have nothing else to do. Almost any money would be well spent if it secured for us the elimination from our midst of the pests of discomfort and disease which arise from mosquitoes. It is only by the close study of their habits and life-history that this desirable end can be secured. Though the aid of general practitioners in various parts of the States is desirable also, the work must be under the direction of a man who has nothing else to do.

PULMONARY DISEASES AMONGST THE QUARTZ MINERS OF BENDIGO, VICTORIA.

By Walter Summons, M.D., D.P.H. (Melb.).

Mining is essentially an unhealthy occupation, seeing that it is carried on in confined spaces that are difficult of ventilation, and also removed from the beneficent effect of the sun's warmth and light. Consequently it has been known for many years that the miners of Bendigo have been an unhealthy class. This paper is the condensed report of a six months' investigation, containing as well the opinions and conclusions arrived at after three years' private practice in the Bendigo district.

The "miners' complaint" of Bendigo is, in all cases, disease of the respiratory organs. No case of ankylostomiasis was found or heard of. My researches support conclusively the outcome of similar investigations in other parts of the world, and the type of the disease is firstly, a pneumoconiosis produced by inhalation of mineral dust; and secondly, a superadded tubercular infection. The lung is very tolerant of the mechanical irritation, but the bacterial infection quickly makes inroads into the damaged tissue with a fatal, and not infrequently early termination.

Statistical.—For the determination of the extent to which phthisis and respiratory diseases prevail amongst the Bendigo miners, the death registers of the district were perused, the search being carried backward for thirty-five years. Prior to this machine rock drills were not in common use, holes for blasting having been made by hand-drills. With the introduction of the machine-drill, which enabled mining to be carried on with greater rapidity, the amount of dust produced underground was largely increased. During the period there has been a progressive and marked increase in the number of miners dying of lung diseases, notably tuberculosis.

Annual Deaths amongst Bendigo Miners, determined in 5-year periods, and estimated as per 10,000 living at all ages.

Year.	Miners' Phthisis (Tuberculous).	Chronic Bronchitis.	Acute Pneumonia.	Total Lung Diseases.	Total Mining Accidents.	All Other Causes.	Total Deaths.
1875–79 ...	48·5	8·0	20·5	77·0	28·5	74·3	179·8
1880–84 ...	56·9	13·1	17·0	87·0	32·8	70·6	190·4
1885–89 ...	80·0	39·3	20·6	139·9	31·3	111·3	282·5
1890–94 ...	84·6	34·4	16·9	135·9	28·4	120·2	284·5
1895–99 ...	102·4	35·6	27·4	165·4	21·9	130·4	317·7
1900–04 ...	100·8	23·0	21·3	145·2	14·7	96·5	256·3
1905–09 ...	109·6	23·0	23·0	155·6	17·6	115·0	288·2
1910 ...	106·6	13·6	27·2	149·6	18·6	114·0	282·2

The death-rate from chronic bronchitis has been estimated in the statistics separately from those due to tuberculosis of the lungs, but I am convinced that a large number of the former should have been certified to as being tuberculous. Acute pneumonia is also tabulated by itself, and its prevalence shows but little variation. The death-rate is in excess of the mortality amongst adult males of the non-mining class (about 27 to 12).

The total deaths from lung diseases have risen from an annual rate of 77 per 10,000 in the first quinquennial period to one of 155·6 in the last. This is largely accounted for by the increase in those certified to as being due to tuberculosis. This disease represented by 48·5 in the first period, now amounts to 109·6 per 10,000 per annum. This increase is overwhelming, and out of all proportion to the annual mortality rate from consumption amongst adult males. The corresponding mortality for Victoria as a whole is less than at any previous time. The mortality rate from tuberculosis amongst miners is at the present time seven and a half times as great as amongst adult males of all occupations in Victoria. This affords ample evidence of the detrimental conditions under which quartz-mining is carried on.

Nature of disease.—The onset of the pneumoconiosis is most insidious, and only after years of work does evidence of the disease present itself in the great majority. (There are, of course, cases of consumption amongst young miners who should never have gone below at all.) Frequently recurring coughs and mild bronchitis attacks may be present for years while the men are still capable of working underground. Sooner or later, however, dyspnœa on exertion becomes felt. This breathlessness increases *pari passu* with the amount of harm done to the lung, till, finally, though constitutionally the man's health is good, he is compelled to cease working. Later on in the case there is wheezing with prolonged expirations, even while resting, while on the slightest exertion there are marked stridor and hurried breathing. This dyspnœa is unassociated with any signs of cardiac incompetence, and is purely respiratory. In no case is the interference with respiration so extreme as to cause cyanosis.

Such, in brief, is the history of the case of pure fibrosis. The general health may be in every way satisfactory. These cases give a high percentage of hæmoglobin, with increase in the number of red corpuscles. A blood count of over five and a half millions is not exceptional with a hæmoglobin percentage of 110.

The great bulk of these cases become subject to asthmatical and continued bronchitic attacks. Repeated examination of the sputum, however, only shows cocci and putrefactive organisms.

On physical examination, diminished chest expansion was evident. In a few cases only was there a tendency to be barrel-shaped, in accordance with which none of the lungs examined were of the large lunged emphysematous type, but there was much coarse emphysema in normal sized or contracted lungs.

Taking everything into consideration, there are no distinctive signs by which one can say that this is a miner's lung, but all point to a chronic irritation of the lung; and the results, in addition to being non-remediable, are, to a greater or less extent, steadily progressive. The average duration of work underground, in the case of eighty-five men who had worked continuously in the Bendigo mines before ill-health necessitated their ceasing work, was twenty-two years.

Associated with the symptoms already given, there is little or no constitutional disturbance. The disease is a purely local one of the respiratory organs, and the symptoms point absolutely to these. Though secondary disorders of the heart and other organs are to be expected, such were late accompaniments, and in every instance subsequent to infection by the tubercle bacillus, and thus resulted from the combined action of both the fibrosis and the tuberculosis. Sooner or later there is progressive loss of weight and weakness, and symptoms suggestive of a more malignant agency at work than mere dust particles. The expectoration may be scanty or profuse and muco-purulent, but microscopic examination, if not the first time, with repeated search, reveals the presence of tubercle bacilli.

Incidence of Tuberculosis.—Though the irritation of the lungs has gone on for years, the onset of symptoms is often remarkably rapid. The question then arises: is this a case of dust irritation of the lungs merely, or is there a superadded infection with the tubercle bacillus. The physical signs do not settle this point. In arriving at the proportion of cases that are tuberculous, sputum examinations were repeatedly made before a negative result was decided on. In addition, the diagnostic use of Koch's old tuberculin was availed of in as many suspicious cases as could be persuaded to come under supervision for a few days.

The dose injected was ·005 c.c., and in a few cases the injection was repeated, giving as much as ·01 c.c. Out of thirteen cases so inoculated, two gave a definitely positive reaction. [Five years ago Calmette's and Von Pirquet were not in vogue.]

Inclusive of the results of autopsies, out of 235 cases, positive evidence of tuberculosis was obtained in 117, that is 50 per cent. Though it is only justifiable to consider a case tuberculous if it is positively proved such, yet the clinical history and physical signs in several other cases left little doubt in my mind that they had been infected, but, at the time of examination, the disease was quiescent.

I am now convinced, after practice in the district, and having seen the subsequent progress of many of the cases, that pleurisy in the great majority of cases is a sign of latent tuberculosis of the bronchial glands. This opinion is strengthened by the findings in three autopsies—on two men dying from accident and one from cancer of the lip. These men had no symptoms of lung complaint save pleurisy, and in all three the presence of tubercle in the bronchial glands was proved by a microscopic examination. The post mortem in a fourth man dying from acute pneumonia presented no pleurisy and no tubercle of the glands or elsewhere.

For these reasons, in my mind pleurisy is the signal of future tuberculosis. This latter may not develop for many years, but all the time the disease is lying dormant in the glands at the root of the lung. When a young miner presents himself with pleurisy, I urge on him the advisability of changing his occupation. When speaking of the proportion of infected cases, I did not take pleurisy as proof of tuberculosis. Undoubtedly the 50 per cent. in which positive evidence of tuberculosis was obtained is a low estimate of the infected cases.

At the present time, all Bendigo miners dying of respiratory disease die of tuberculosis, with the exception of acute pneumonia. This statement is based on the final ending in forty-eight consecutive cases, the only exceptions being that of two men who died of acute pneumonia. In twenty-two of these deaths, the presence of tuberculosis was confirmed by a post-mortem examination. It is conceivable that there may be cases that progress as a pure fibrosis with recurring attacks of bronchitis and development of emphysema to such an extent as to cause heart failure. I have, however, not seen any such case. Tuberculosis is so prevalent that such susceptible cases become infected unless carried off by some intercurrent disease as Bright's disease, typhoid fever, and such like.

In the Transvaal Medical Society's Report on Miners' Phthisis, published 1903, and in an address by Sir Thomas Oliver, on Miners' Phthisis, printed in the *British Medical Journal*, 1905, the disease is regarded as almost non-tuberculous, whereas Dr. Haldane looks upon Miners' Phthisis in Cornwall as being mainly of an infective character. Dr. Cumpston is here to speak of the disease amongst West Australian miners. The inference to draw from these almost contradictory opinions, and the evidence obtained from cases at Bendigo, is that the tubercular infection depends on local conditions. If the disease is rare in the community, the chance of infection is diminished, and *vice versâ*.

Pathology.—In treating of the morbid conditions in the lungs, the pathology is based on the evidence obtained from twenty-two autopsies; in the case of many of these I have a full clinical history as well. The essential pathology of miner's phthisis at Bendigo is a fibrosis of the lungs, with an associated chronic irritation of the bronchial tubes, consequent upon which coarse emphysematous changes ensue, and sooner or later infection with the tubercle bacillus takes place, with the resulting development of tubercles, caseation, and formation of cavities. The tuberculous process is somewhat altered from the fact that the tissue affected is not normal, but fibrotic lung tissue.

In microscopic sections of the lungs and the bronchial glands innumerable jagged, opaque, and some translucent adventitious particles are seen. The translucent ones are the flaky quartz, and the opaque are from the hard Silurian bed-rock. Professor Masson kindly incinerated a pair of pigmented lungs for me, and the ash contained 40·5 per cent. of silica and 33·45 per cent. of aluminium. The presence of aluminium is altogether abnormal, while the silica is present in three times the amount normally present in the lung tissue according to Hammarston's analysis. The particles correspond in shape and size and appearance to those found suspended in the mine air, and are seen to lie, in an early case, more concentrated in the lymphatic spaces of the lung. In the more advanced cases the distribution is more uniform, and, to the naked eye, the lung tissue is an inky black colour.

The silicious particles being deposited in the tissues bring about, on account of their jagged edges, a low, chronic inflammation, with the production of formative cells and fibrous tissue, which envelops the irritating particles. However, with the development of this fibro-connective tissue, there is no fresh formation of new blood-vessels, while the existing ones in many places become occluded. Thus the newly-formed tissue interferes with the respiratory processes, and, being of low vitality, has small germicidal powers.

Emphysema, bronchitis, dry pleurisy, sooner or later develop, with increased embarrassment in respiration and finally tuberculosis of the lungs. The site of the infected area is usually apical as in ordinary phthisis. The infection is chronic and the necrotic process slow. The tuberculous nodules infiltrating the pigmented tissue, not replacing it, are likewise pigmented, and the "black spit" that persists, even after the miner has ceased work underground, is due to the presence of this softened lung tissue in the expectoration.

Etiology.—Passing briefly over the etiology, the excessive mortality amongst the Bendigo miners being the result of lung disease, the cause must be looked for in the vitiation of the air.

The mines are not adequately ventilated, and are becoming less so as their depth increases. The CO_2 and other gaseous impurities are not in sufficient quantity or kind to have great detrimental effect; the dust particles must be considered as the potent cause. The air is densely charged with them. They are found constantly embedded in the lungs, and, even after many years, present the same appearance as when first inhaled. On incineration, the dust was regained from the lungs, and by chemical analysis shown to be of the same nature as the country rock of the Bendigo mines.

The other sources of air vitiation are but adjuvant causes. The oppressive heat of the mines does not seem to be highly injurious; probably greater harm results from the sudden changes of temperature experienced on ceasing work and going to the surface. Also, in common with all other depressing occupations, the craving for stimulo-sedatives amongst miners is abnormal.

Prophylaxis.—The disease is thus seen to be non-remediable, hence the greatest attention should be paid to the prophylaxis. This can be briefly said to consist in the absolute prevention of dust by the use of water jets and sprays, and in the improvement of the mine ventilation. By actual estimations the dust has been removed by thorough spraying to the extent of 99·7 per cent. of the amount present in the mine air before the spraying.

Miners' phthisis is by far the most important of the so-called trade diseases in Victoria, and its great prevalence at Bendigo is unwarrantable.

MEDICAL INSPECTION OF SCHOOLS IN QUEENSLAND.

By Eleanor E. Bourne, M.B., C.M. (Syd.).

Systematic medical inspection of school children was first officially recognised as a necessity in Queensland in 1907.

Such alarming reports as to the state of the eyes of school children in the western districts had reached the ears of Members of Parliament, that is was deemed advisable to arrange for a special inquiry. Dr. Taylor accordingly made an extensive survey of the affected country, his efforts being directed almost entirely towards the school children. Of the persons examined, 90 per cent. were found to be more or less affected with unhealthy lids, and a proportion of these with more serious troubles. Various recommendations were made by Dr. Taylor, and a more or less effectual attempt has been made to carry a few of these into effect, by means of the local hospitals and the head teachers of schools.

When the idea of a permanent medical inspection of schools was first mooted, it was largely with the object of inquiring into the working efficiency of the methods in use for dealing with these eye troubles. With Dr. Elkington as Commissioner of Public Health, however, the matter was not likely to rest between these narrow limits. By lectures and speeches, he aroused in people's minds the recognition of the need for a broad and comprehensive scheme, to touch every point of the school child's life.

My own appointment, as Medical Inspector, acting under the direction of the Commissioner of Public Health, took effect from 1st January, 1911. As some little time was occupied in formulating the scheme of inspection, and as the fly season, when eye troubles are worst, was then practically over, it was thought best to postpone the visit to the West, until the next fly-season began—about October; consequently, the work, so far, has been performed in Brisbane and Ipswich, and broadly resembles other medical inspection work in large towns. The time occupied in it has been very brief, and I must therefore ask your indulgence for presuming to read a paper on a rather small number of children examined.

The staff consists at present of one medical inspector, one school nurse, and one dental inspector; the Commissioner of Public Health has undertaken the general direction of the staff, which is attached, however, to the Department of Public Instruction. The medical inspector examines all the children in the schools systematically, the details being entered on the front of a card, as shown below; symbols are used as far as possible in filling up the card items. On the back of this card there is space for six re-examinations, and for special examinations and remarks.

DEPARTMENT OF PUBLIC INSTRUCTION, QUEENSLAND.

School: *Valley* (*Infants*)
1. Name: *A. H.*
2. Place of Birth: *Brisbane.*
3. Parent's Occupation: *Draper's assistant.*
4. Age last birthday: 5.
5. Class: I.
6. Disease History—
    Measles: +
    Whooping cough: —
    Diphtheria: —
    Scarlet fever: —
    Others: —
7. Physique: *C.*
8. Nutrition: *B.*
9. Clothing: *A.*
10. Intelligence: *B.*
11. Speech: *B.*
12. Cleanliness—Head: *A.*
    Body: *A.*
    Clothing: *A.*
13. Conjunctivæ: ✓
14. Corneæ: ✓
15. Squint: —
16. Mouth breathing: +
17. Adenoids: + T + +
18. Ear discharge: —, *Earache,*
19. Teeth: C. 3
    Carious molars T. 2 or P.o.
    Others T. 3 or P.o.
20. Skin: ✓
21. Scalp: ✓
22. Glands: +
23. Heart: ✓
24. Lungs: ✓
25. Anæmia: +
26. Physical Defects: *Nil.*
27. Others: *Nil.*

Height.	Weight.			L.V.		R.E.	L.E.	Others.	I.E.P.
		N.	D.	N.	D.				
Inches. $40\frac{3}{4}$	Pounds. $34\frac{1}{2}$	....	$\frac{6}{6}$	...	$\frac{6}{6}$	$\frac{6}{18}$	$\frac{18}{18}$	...	...

Notification to parent : *Adenoids and enlarged tonsils. Deafness.*

Advice to Head Teacher : *To sit near front, on teacher's left.*

(Signed) Eleanor E. Bourne,
(Date) *6th April,* 1911. Medical Inspector of Schools.

A Medical Register is kept at the school, and in this the teachers are required to note, in the intervals between the visits of the medical inspector, any child to whom they consider medical attention should be drawn, and the reasons for their opinion. In this book I also enter, for easy reference, the names of children who are under special attention of any kind from the teacher, with full directions. The following conditions are included :—Eye strain, instructions being given as to position in class or maintenance of a sufficient reading and writing distance; deafness, these cases being also placed in the most advantageous position; heart disease, directions being given as to mental strain, and the avoidance or modification of physical exercise; nervous affections, including fatigue after illness, and nervous instability, as in chorea and exophthalmic goitre.

In the case of children whose educational progress is being interfered with by their physical condition, a notice is sent to the parents, advising them to take the child to a doctor. These include, for the most part, the following:—adenoids, when accompanied by mouth-breathing or ear-discharge or deafness, or when producing physical defects such as pigeon breast, or even when present in small amount, if the child is of markedly inferior intelligence; greatly enlarged tonsils; defective vision, $\frac{6}{12}$, or $\frac{6}{9}$ if accompanied by eyestrain; heart disease, of which the parents are very frequently in ignorance; marked anæmia; deafness more than $\frac{15}{18}$, the test being a whisper at 18 feet; and chorea. Frequently notices have to be sent advising parents to let the children wear wool next the skin, in heart diseases, for example; or to leave the bedroom window open at night.

For purposes of exclusion for infectious diseases, a notice of a different colour and a more summary tone is sent, which states the period for which exclusion is necessary.

The school nurse usually visits the schools with me, and assists by weighing and measuring the children, examining their heads, and doing a good deal of the routine eye-testing. Another of her duties is to visit the homes of children, who have been recommended for medical treatment, after the lapse of a certain interval. In this way, we are enabled to gain much information in an informal way, and to correct many erroneous idea about the aims of medical inspection; the percentage of children who have been treated on the recommendation of the Medical Inspector can be ascertained, and parents who have neglected to seek treatment for the children may be urged to action.

The Dental Inspector makes a routine examination of every child, the result being recorded on a chart, a duplicate of which is sent to the parent.

Up to the end of June, 1911, 3,068 children had been examined in Brisbane, and since then 344 have been examined in Ipswich.

The number of children suffering from adenoids in marked degree was 822, or over 26 per cent.

The number with defective vision, to the extent of $\frac{6}{12}$ or worse, was 116, or about 3·8 per cent. This is much less than the percentage I expected to find. The infrequency of eye defects is not to be accounted for by the superior construction of Queensland schools; for school architects in Queensland have given just as little attention to these matters as in any other part of the world. Rather must we attribute this result to the brilliant sunshine which overcomes all obstacles, and partly to the Queensland climate, which attracts young people to live in the open air, and not to keep their noses overmuch glued to their books. In the schools themselves, children are not kept always in the same class-room, but are moved about, often to the verandah or shed, so that if bad conditions exist, they are not allowed to act continuously. Consequently, one is not as a rule able to sheet home the blame to the class-room, where visual defects are found particularly prevalent in certain classes of a school, though one would sometimes like to do so.

A table is given which shows the different degrees of visual defects found. It will be noticed that, when one eye is affected, the left is deficient far more frequently than the right. At first I was afraid that some error might be responsible for this, but the same result has been recorded by other observers. Girls are noticed to be affected with visual defects more than twice as frequently as boys, and this has also been noticed by others, sedentary habits being largely responsible.

The heart is examined in every case, with the result that 61 children with weak hearts were discovered, 34 of these having really serious defects. Almost half of them give a history of rheumatic fever; but in many cases, neither parents nor children seem to be aware of the condition, some of the children giving vague histories of "growing pains" or sore throats.

As a good deal of this examination was performed in the summer, a rather large proportion of children (about 17 per cent.) showed the pallor and lassitude which has to serve as a rough indication of anæmia, owing to the short time available for inspection. Only a few of these, however, were notified to the parents.

TABLE I.

(*a*) Table showing the effect of physique, nutrition, and various physical defects upon a child's intelligence.

Intelligence.	No.	1 Physique.			2 Nutrition.			3 Adenoids.			4 Defective vision.		5	6	7	8	9	10
		A	B	C	A	B	C	* Marked.	Slight.	Removed.	* $\frac{6}{12}$ or worse.	$\frac{6}{9}$	Deafness.	Weak hearts.	Enlarged glands.	Anæmia.	Physical defects.	Mental deficiency.
A ...	1,328	741	417	170	867	373	88	290	384	38	46	111	53	20	278	151	270	0
B ...	1,414	661	495	258	820	479	115	401	423	54	56	128	74	30	340	298	442	0
C ...	326	109	128	89	158	114	54	131	81	19	14	26	24	11	104	85	131	7
Total ...	3,068	1,511	1,040	517	1,845	966	257	822	888	111	116	265	151	61	722	534	843	7

(*a*) Table of percentages showing the effect of physique, nutrition, and various physical defects upon a child's intelligence.

Intelligence.	No.	Physique.			Nutrition.			Adenoids.		Defective vision.		Deafness.	Weak hearts.	Enlarged glands.	Anæmia.	Physical defects.	Mental deficiency.
		A	B	C	A	B	C	* Marked.	Total.	* $\frac{6}{12}$ or worse.	Total.						
A	1,328	55·8	31·4	12·8	65·3	28·1	6·6	21·8	50·8	3·5	11·8	4	1·5	20·9	11·4	20·3	0
B	1,414	46·8	35	18·2	58	33·9	8·1	28·4	58·3	4	13	5·5	2·1	24	21·1	31·2	0
C	326	33·4	39·3	27·3	48·5	35	16·5	40·2	65	4·3	12·3	7·4	3·4	31·6	26·1	40·2	2·1

NOTE.—In this table the columns marked with an asterisk contain conditions which are interfering with the child's intellectual progress. "Deafness" does not include the deafness accompanying adenoids. The physical defects are exclusive of those incidental to the presence of adenoids. The numbers represent individual defects, not individual children in columns 3 to 10.

TABLE II.—Analysis of Visual Defects.

Degree.	One eye affected.		Both eyes affected.	
	Right.	Left.	Right.	Left.
$\frac{6}{9}$	67	92	121	114
$\frac{6}{12}$	4	11	21	21
$\frac{6}{18}$	1	15	12	13
$\frac{6}{24}$	0	6	9	12
$\frac{6}{36}$	2	3	4	4
$\frac{6}{60}$	2	1	1	3
Less	4	4	1	2
	80	132	169	169
Total ...	212		169	

Total number of visual defects = 381.

Altogether, 31·71 per cent. of the Brisbane children were found to be defective to an extent interfering with their educational progress.

Table I shows the association between physical and intellectual efficiency; 27·3 per cent. of the children with poor intelligence have also poor physique, while only 12·8 per cent. of the children with good intelligence have poor physique. For the sake of argument, one sometimes wishes the relationship were even closer; it is somewhat disconcerting to a school hygienist to be faced by a weedy weakling at the head of the class. The reason for such an anomaly probably is that these children, cut off from healthy physical rivalry by their infirmities, take all their chances of success on the spiritual and intellectual side. One could wish that the success might continue through life, but, unless based on a sound physical basis, it is often only too transitory.

As we should expect, the children of poor intelligence show a large percentage of adenoids, also of weak hearts; the association of visual errors with dulness is, however, obscured by the fact that hard study in an intellectual child may actually develop such errors.

Interesting variations in the frequency of physical defects are found in different schools. The schools examined so far may be described as (1) a better-class old-fashioned city school, drawing a large proportion of children from good suburban homes; (2) a school whose pupils come from low-lying, damp, and poor houses; (3) a new school, well lit and drained, but rather near the sea; (4) a school on a well-drained, gravelly hill site, the pupils coming from similarly situated homes; (5) a school in a limestone town, inland and fairly high.

In (1) adenoids were not very prevalent, but defective vision was very common; in (2) adenoids were markedly prevalent, and enlarged glands were found in almost every one of the younger children, eye defects were rather frequent also; in (3) adenoids were fairly frequent, but eye defects were hardly seen; in (4) eye defects were common, adenoids not very common, weak hearts were rather frequent; in (5) adenoids were remarkably infrequent, not quite 8 per cent. being found, teeth were much better than usual, vision was good, weak hearts were rather frequent. Table III and Graph I give the percentage of physical

GRAPH I.—SHOWING PERCENTAGE OF PHYSICAL DEFECTS ACCORDING TO CLASSES.

Class	I.		II.		III.		IV.		V.		VI.	
	Girls.	Boys	Girls.	Boys.	Girls.	Boys.	Girls.	Boys.	Girls.	Boys.	Girls.	Boys.
Total No. in Class ...	292	330	264	296	232	304	252	294	224	288	101	191
Adenoids I.E.P.	100	139	68	102	65	96	51	68	36	48	9	37
„ Total	179	230	156	199	128	194	132	165	93	134	32	90
Defective Vision I.E.P.	10	10	8	8	9	12	9	4	17	8	11	7
„ Total	26	37	42	25	33	33	35	25	47	19	25	23
Anæmia I.E.P.	1	0	2	0	7	0	2	1	3	0	0	0
„ Total	26	45	33	41	42	53	24	42	53	66	28	40
Others I.E.P.	15	3	5	11	6	7	6	9	1	11	0	3

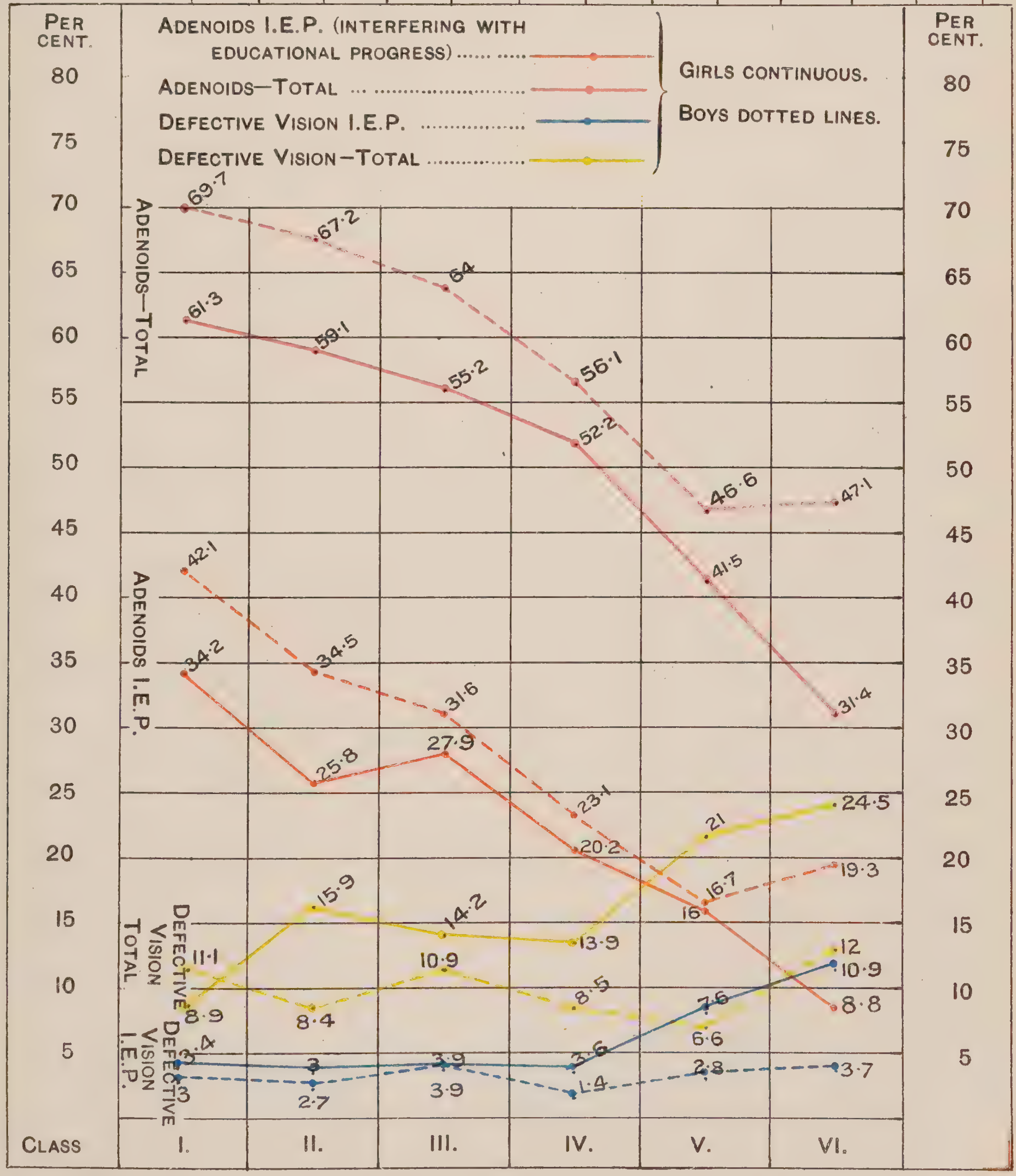
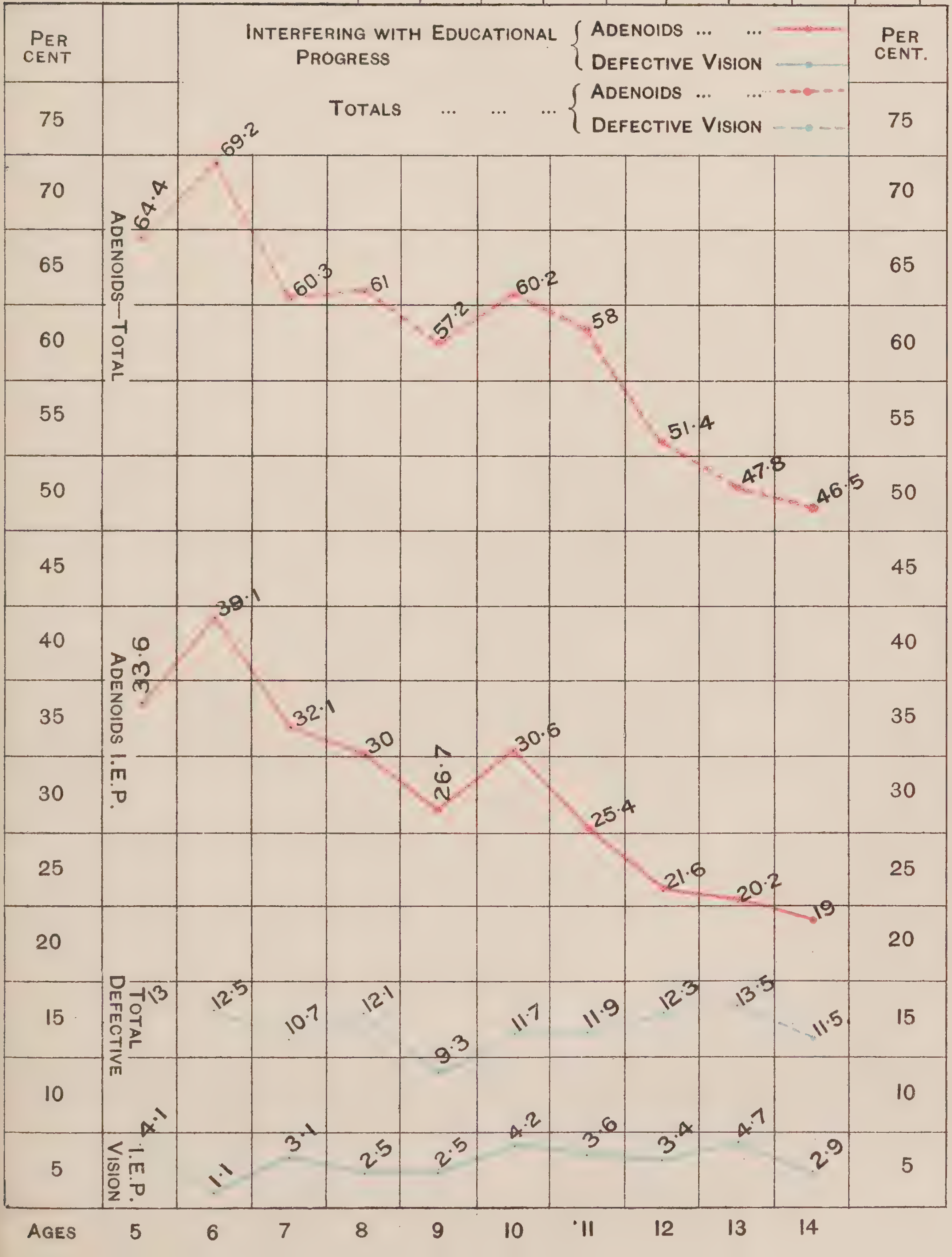


Table III.—Showing Number of Physical Defects according to Classes.

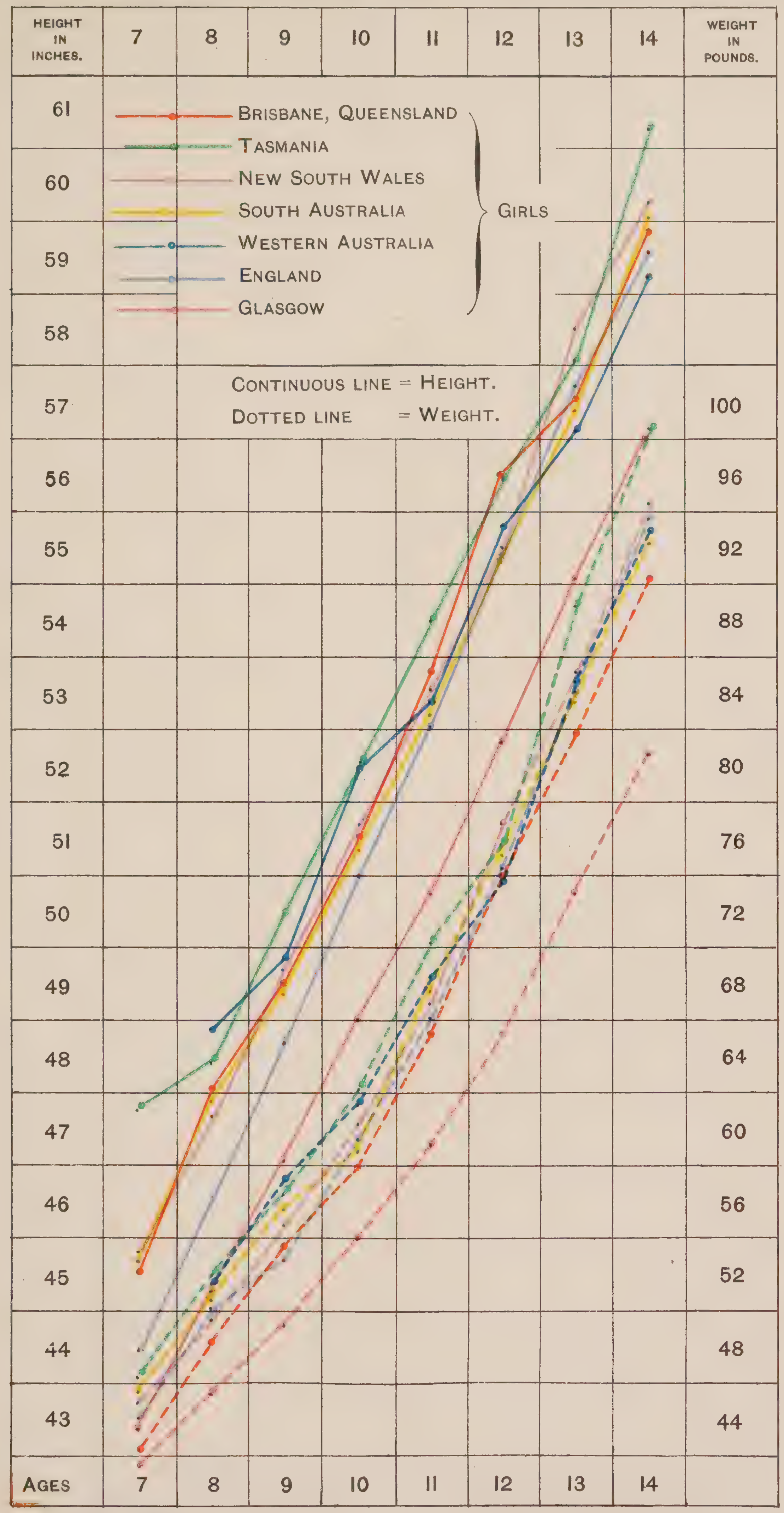
GRAPH II.—SHOWING PERCENTAGE OF PHYSICAL DEFECTS ACCORDING TO AGE.

Table IV.—Showing Number of Physical Defects according to Age.

AGES	5	6	7	8	9	10	11	12	13	14
TOTAL NO. EXAMINED AT DIFFERENT AGES... ...	146	263	262	323	367	359	386	407	362	174
ADENOIDS I.E P.	49	103	84	97	98	110	98	88	73	33
TOTAL	94	183	170	197	210	216	224	209	173	81
DEFECTIVE VISION, I.E.P.	6	3	8	8	9	15	14	13	17	5
TOTAL	19	33	28	39	34	42	46	51	49	20

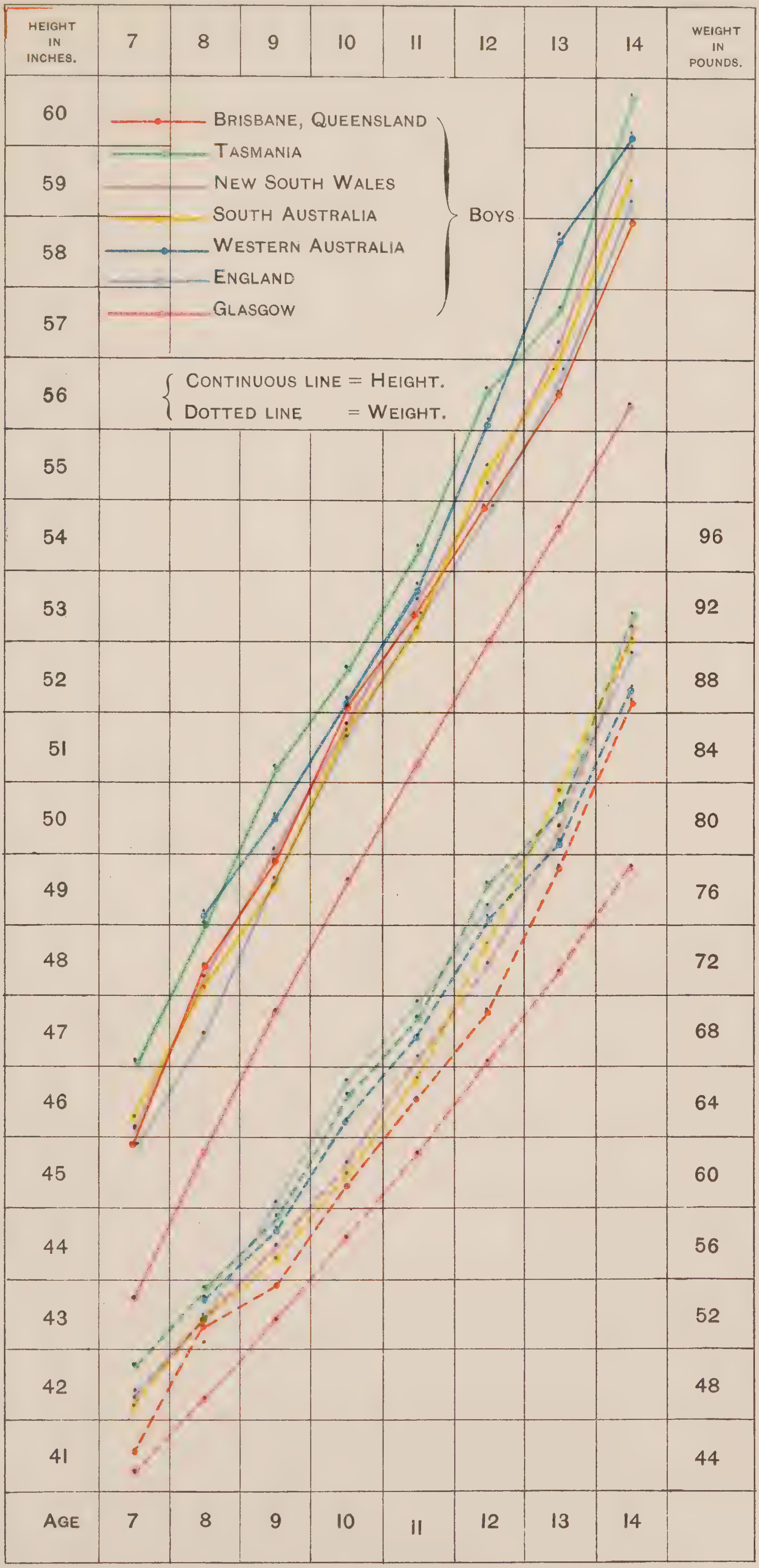


GRAPH III.—COMPARING HEIGHTS AND WEIGHTS OF BRISBANE GIRLS WITH THOSE OF ENGLAND, GLASGOW, AND OTHER AUSTRALIAN STATES.



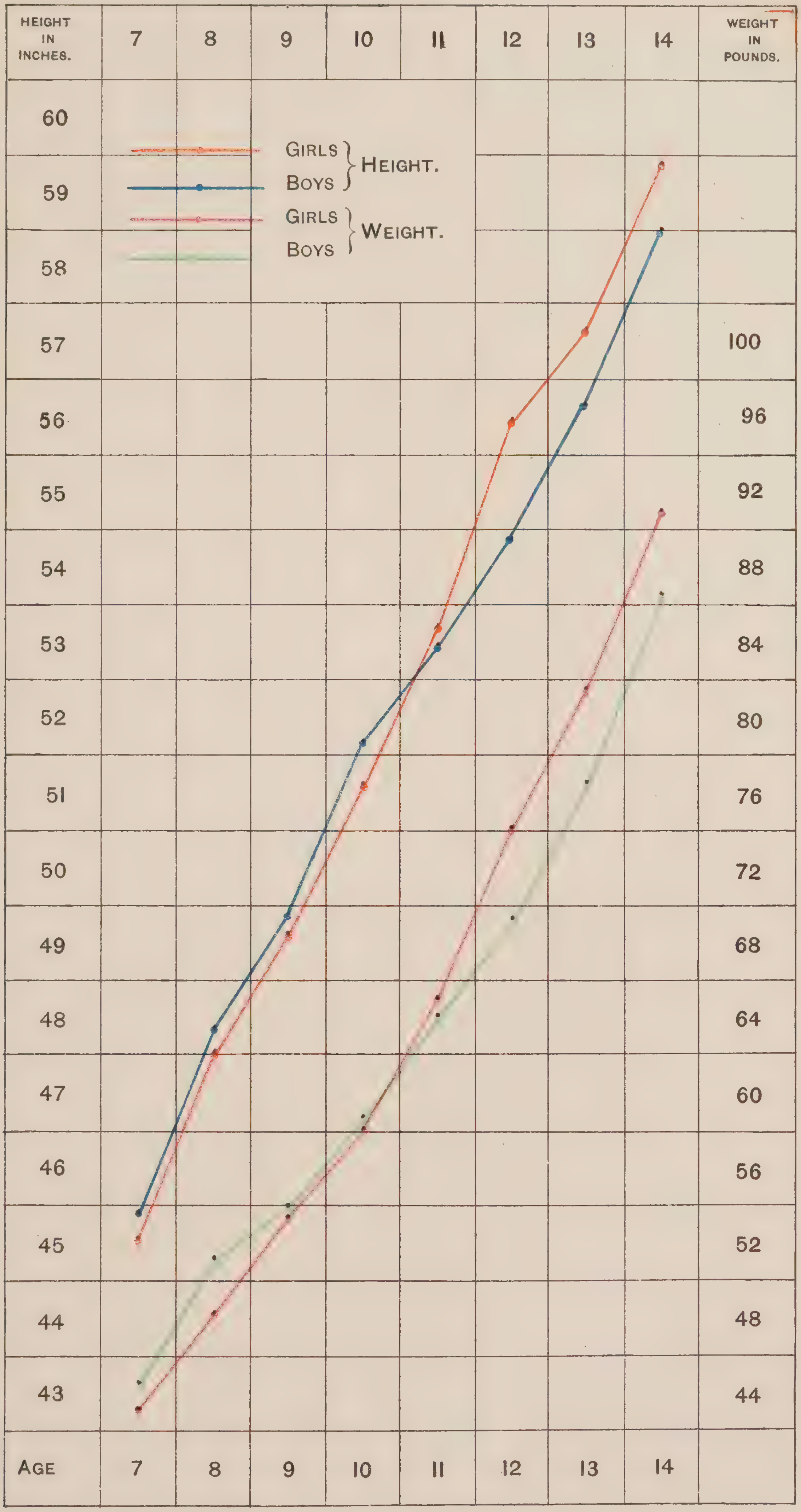
Note.—English Children wore Boots during the Examination.

GRAPH IV.—COMPARING HEIGHTS AND WEIGHTS OF BRISBANE BOYS WITH THOSE OF ENGLAND, GLASGOW, AND OTHER AUSTRALIAN STATES.



NOTE.—ENGLISH CHILDREN WORE BOOTS DURING THE EXAMINATION.

GRAPH V.—COMPARING HEIGHTS AND WEIGHTS OF BOYS AND GIRLS IN BRISBANE, QUEENSLAND.



defects found, arranged in classes. Defective vision is seen to increase enormously in the upper classes, especially among the girls. Adenoids appear to be much more prevalent in the lowest classes; this is because I notify the younger children more frequently, as I think that, if the removal of adenoids is to do good, it should be done before physical defects, such as pigeon breast, have a chance to develop.

Table IV and Graph II show the physical defects arranged according to age. In this the increase in the percentage of visual defects does not show for advanced ages; indeed there is a fall, probably because these children leave school at 12, on account of the discomfort caused by their condition.

The height and weight of Brisbane girls and boys is compared with those of children in other Australian States and of England and Glasgow in two graphs. In height they compare well with other children, and in weight they are only slightly inferior; and it must be remembered that the examination was done in summer, when the lightest of clothes are worn, and that a light weight is rather an advantage in a hot climate.

The height and weight of boys and girls is compared in Graph V, the girls showing the advantage of a somewhat early pubertal development.

Of the physical deformities found, the vast majority were due either to slackened muscles, or to the effect of adenoids. In the former class may be placed lateral curvatures, 77; round shoulder, 192; inequality in height of shoulders, 32; and flat foot, 78. In the latter class come high palate, 150; many of the chest deformities, 211 altogether; and others, such as prominent upper teeth, 23. Other deformities are congenital, such as hernia, 6 cases, stumps of limbs, 2 cases. Hernia was only asked for, not looked for, so the numbers are not to be regarded as correct. Some cases of paralysis, 7 altogether, are reminders of the epidemic of infantile paralysis which occurred about seven years ago. Birth palsy, meningitis, and lead poisoning account for the 4 other cases. Rickets was believed to be the cause of chest deformity in 5 cases.

It would be idle to pretend that medical inspection of schools gives an absolutely true picture of the amount of syphilis which is present in a community; there are so many factors which cannot be taken into account in school inspection work. For one thing, it is impossible to get the family history, upon which we rely so much in the diagnosis. Another point is that a very large number indeed of syphilitic children die in infancy, and never arrive at school age. But even taking these matters into consideration, the number of cases found which suggest congenital syphilis, 16, does not seem to point to a very wide-spread infection in Queensland at any rate. Most of these showed pegged teeth, some showed other signs, depressed nasal bridge or pigmented symmetrical scars. No cases of interstitial heratitis were met with in the schools examined, which do not nearly cover the whole of Brisbane.

In view of the enormous number of physical deformities due to ill-developed muscles, it is a pity more serious attention is not paid to physical exercise, especially among the girls.

I think there is no reason why, up to the age of 12, when Commonwealth Military Regulations claim the boys, both boys and girls should not be exercised together, when the languid movements considered lady-like in many girls' schools would become things of the past. Exercises without cumbrous apparatus are the ones most likely to be generally used, and so would be most valuable. The syllabus prescribed by the Board of Education, London, appears to give a very thorough training. Swimming and games must, of course, be highly recommended, but in large schools, the children who indulge in them are apt to be those of a better class; the poorer children, who need the exercise more, evade it by some means; while the school pats itself on the back, and says, "Look and see what we are doing for the development of the children."

Long desks and unbacked forms have received so many blows on behalf of the children, that one hardly needs to refer to them as sources of curvature. Bags are other fertile causes; the children for some reason—fashion probably—prefer to hold them so that their weight acts to the greatest advantage from the tip of the shoulder; consequently, inequality in the height of shoulders is fairly common. Shoes are also to be blamed for many cases of flat foot. Graph VI shows the maximum differences of height of children in any one class, all of whom sit at forms and desks of practically the same size; 19 inches is the difference in one case between the tallest and shortest boy, and affords ample room for the development of a curvature.

It may be noticed that in Queensland, beyond the records of height and weight, anthropometry as yet receives scant consideration. The country is so large and the aims so utilitarian, that it has been deemed best, for some time at any rate, to confine our efforts to removing the obvious physical defects, which are seen to be working present havoc. These apparently simple aims will keep us busy yet for some years.

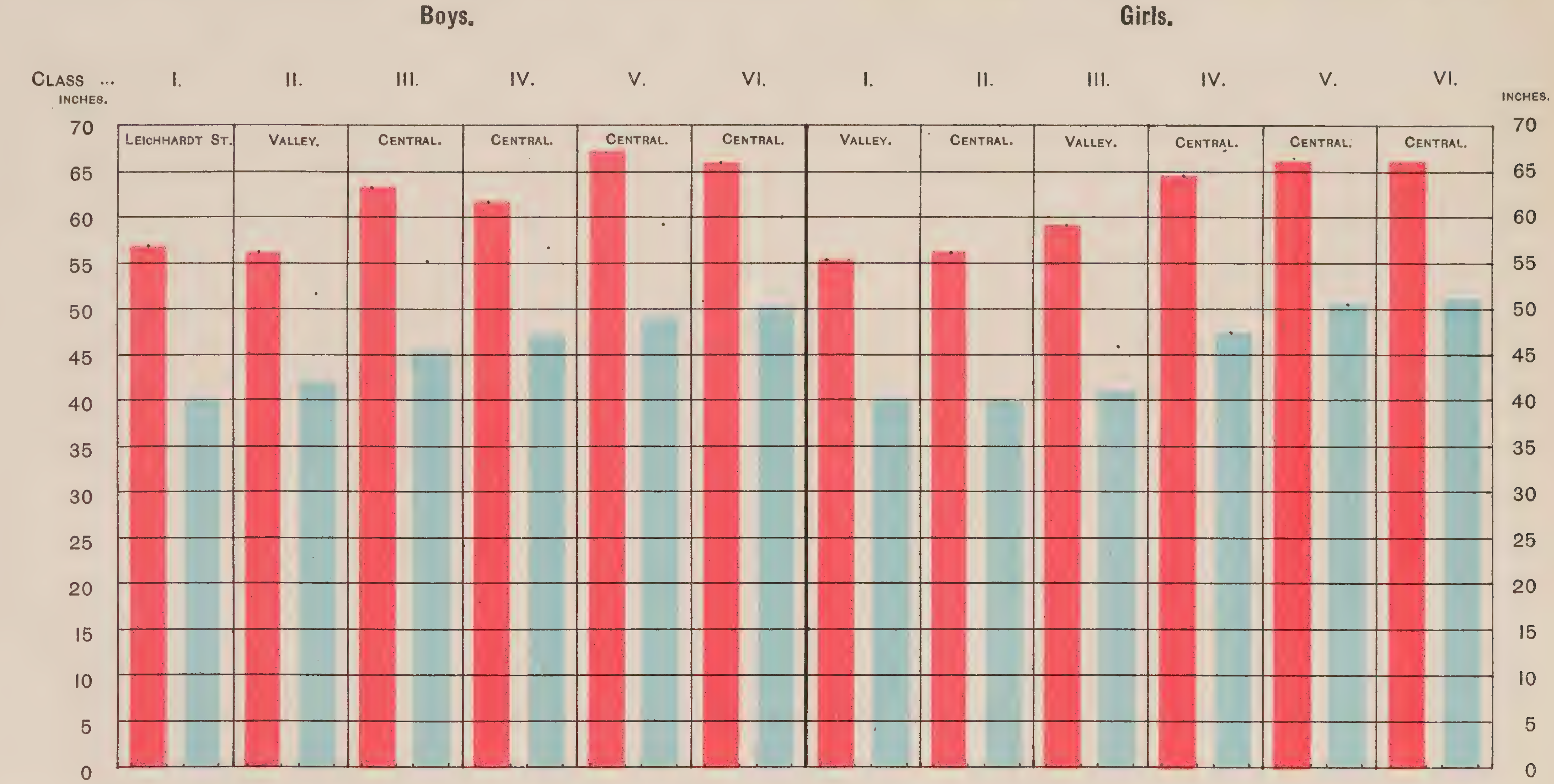
Besides the mere inspection work, the scheme of medical inspection in Queensland is largely educational. Lectures are given to the teachers in metropolitan schools once a week, as well as talks to the teachers at each school to demonstrate the significance of the more obvious physical defects. Talks are given to the children at the close of a school visit; and later, it is hoped to arrange a series of lectures to parents, which it is expected will be very valuable in the country districts, for instance, with regard to the prevention of eye diseases.

The influence of school buildings also received attention, and reports are made to record obvious defects in each school examined.

Certain defects are very frequently noticed. A lack of attention to the needs of infant teaching is one of these; if economy is necessary in the matter of space or expenditure on furniture, the infants' department bears the brunt of it. Other defects are the almost universal use of long desks and unbacked forms, and the inadequate area of the playground in certain metropolitan schools.

It is hoped that medical inspection, when complete, will be of great benefit in educating both parents and children in the methods of preventing certain diseases, which are rather prevalent in Queensland. These diseases vary according to the varying conditions in different parts of the country. They are eye diseases, the most troublesome being trachoma, in the dusty, fly-ridden western plains; anchylostomiasis in certain of the moist coast areas; and mosquito-borne diseases such as filariasis and malaria, mostly along the tropical coast line. All of these are amenable to enlightened methods of sanitation and hygienic living; consequently, all of them should be attacked, on one side at any rate, through the educational system.

GRAPH VI.—SHOWING THE MAXIMUM DIFFERENCES IN HEIGHT IN ANY ONE CLASS, BRISBANE SCHOOLS EXAMINED.



	Boys.						Girls.					
CLASS ...	I.	II.	III.	IV.	V.	VI.	I.	II.	III.	IV.	V.	VI.
	LEICHHARDT ST.	VALLEY.	CENTRAL.	CENTRAL.	CENTRAL.	CENTRAL.	VALLEY.	CENTRAL.	VALLEY.	CENTRAL.	CENTRAL.	CENTRAL.
MAXIMUM	56¾ IN.	56½ IN	63¼ IN.	62 IN.	68 IN.	66½ IN.	55½ IN.	56½ IN.	58¾ IN.	64¾ IN.	66¼ IN.	66¼ IN.
MINIMUM	40 ,,	42¼ ,,	45¼ ,,	46½ ,,	49 ,,	50 ,,	40 ,,	40 ,,	41½ ,,	47½ ,,	50½ ,,	51¼ ,,
DIFFERENCE	16¾ IN.	14¼ IN.	18 IN.	15½ IN.	19 IN.	16½ IN.	15½ IN.	16½ IN.	17¼ IN.	17¼ IN.	15¾ IN.	15 IN.

One thing I sincerely hope medical inspection will do in Queensland—that is to help to dissipate the idea that the climate makes the country essentially unfit for white people to live in. It is in childhood, before the body becomes a toy of various confusing pathological cross-currents, that such a point may best be judged. It is seen from these graphs that the height and weight of Queensland children are nothing to be ashamed of, and the percentage of physical defects compares favourably with those of other countries. Let us hope that medical inspection may do its part in assisting this country to the top of the wave of prosperity which appears to be coming.

SMALL PRIVATE SEPTIC TANK INSTALLATIONS.

By T. W. Sinclair, M.D., B.S., D.P.H.

The introduction and general adoption of the water-carriage system for the removal of wastes from dwellings have proved of untold benefit from the standpoint of public health and sanitation generally. Such being the case, opportunity to secure what are virtually almost equal advantages has arisen for persons dwelling in districts where there is not a system of sewerage available, through the agency of biological methods, or more commonly described septic tanks. The decided and real advantages provided by a water-carriage system have no doubt been largely responsible for the extensive adoption by private individuals of small septic tanks in the unsewered areas of the metropolitan districts of Sydney, but a further inducement has been the natural configuration of the area with its extensive length of foreshore, and the opportunity which is thereby provided for the disposal of tank effluents by discharging them into tidal waters below low-water mark. It has been a universal observation that the incidence of typhoid fever has always undergone a considerable reduction where water-carriage system of removal has replaced the conservancy method of pails; so it is possible that the use of private septic tanks for dwellings under a proper system of control and construction may tend more or less in the same direction.

Installations intended for the treatment of sewage from individual dwellings, through the agency of biological methods, began first to be used in the metropolitan districts of Sydney about twelve years ago. Since then they have continued to be extensively adopted, until, at the present time, they amount approximately to about 450 in number.

In earlier years, local authorities possessed no proper legal powers for controlling the construction and use of such methods of sewage disposal by private persons, and no rules affecting the erection and position of these installations could be enforced. As a result of the absence of control chiefly, and to a less extent owing to the want of definite ideas as to the nature of the processes involved in these methods, those installations constructed in the earlier years consisted simply of a single covered water-tight compartment of liberal dimensions, into which the sewage was discharged, and from which it escaped by an outlet pipe into the earth or tidal water.

The increasing extent to which private persons began to avail themselves of this method of sewage disposal forced itself upon the notice of local authorities, and in the year 1902 a set of by-laws was drawn up by the

Department of Public Health, on the suggestion of the Medical Officer of Health (Dr. W. G. Armstrong), for voluntary adoption by local authorities which required powers for dealing with such installations. These by-laws were adopted by most of the local authorities in the metropolitan unsewered areas, and complete control of private septic tanks in their respective districts was thereby secured to them. Subsequently the by-laws were incorporated in Ordinance No. 45 of the Local Government Act, 1906, and became applicable throughout the whole of the State of New South Wales, outside of the City of Sydney.

The by-laws are as follow :—

Method of Disposal of Night-soil.

5. Unless and until otherwise directed by the Council, all night-soil shall be disposed of by burial in the earth in the manner described in this ordinance, or by treatment in a septic tank of a design and character approved by the Board of Health, or by discharge into the open sea.

Septic Tanks prohibited after Councils has directed Connection with Sewerage System.

26. (*a*) A septic tank shall not be constructed, and if already constructed shall not be continued, on any premises after a date on which the Council or any authority in the exercise of statutory powers shall have required such premises to be connected with a sewerage system, if such is, in the opinion of the Council or of such authority, available.

Septic Tanks shall not be constructed without permission.

(*b*) No septic tank shall be constructed on any premises without permission of the Council first obtained.

Persons desiring to construct Septic Tanks to apply to Council.

(*c*) The owner of premises on which it is desired to construct a septic tank shall, before commencing such construction, apply in writing to the Council for permission to construct it. Such application shall be accompanied by—

Complete plans and specifications of proposed works; and a block plan, to scale, of the premises and of the adjoining public ways and places, on which the proposed position of the septic tank is accurately shown; and

A statement of the way in which it is proposed to dispose of the effluent waters from the septic tank;

and the Council may in their discretion grant or withhold permission.

(*d*) If for any reason a septic tank become at any time a cause of nuisance, or of danger to health, the Council may, by notice in writing, direct the owner of the premises to remove it, and the occupier of the premises to cease from using it forthwith. No person shall refuse or neglect to comply with the terms of any such notice.

Since the by-laws came into force, generally it has been the practice in the metropolitan districts for local authorities to request the advice of their medical officer of health in regard to the granting of permission for the construction of septic tanks on private premises, and on the suitability of the plans submitted for the purpose. I need hardly add, that this procedure has led in some ways to a uniformity in regard to certain important matters of construction of such installations. In advising on such applications, an inspection of the site of the proposed installation has been made to determine in the first place its suitability. The conditions on which suitability of site depend vary considerably in different cases.

Where opportunity exists for sewage, after treatment, to be discharged into tidal waters below low water, there is little or no difficulty. However, where such a method of discharge is not available, more care is required in determining suitability of site so that the disposal of the tank effluent may be effectually provided for. For effective disposal, an adequate area of land belonging to the premises, or which can be made available for the purpose, is necessary.

It is more or less impossible to lay down any standard which might be regarded as a minimum area that is requisite, and each case must be dealt with according to circumstances. A personal inspection of the site is a matter of importance, for the nature of the soil, fall of the land, and the presence of outcropping rock, are all intimately concerned with the proper and effective disposal of a tank effluent. Most favourable conditions are found where there is deep sand or sandy loam; while the reverse occurs where clay or rock predominates or is present to any considerable extent, that will interfere with the porosity of the soil and its absorbing powers for moisture.

Where sites are flat or have very little fall, and the subsoil is of a clayey nature, it becomes necessary for the effluent to be collected in a deep water-tight compartment, from which it must be raised daily by pumping either by hand or by other means, for its effective disposal in the superficial layers of soil. Unless strict attention is paid to the regularity of the pumping, nuisance may arise, and this constitutes a difficulty actually met with in practice in the type of sites just mentioned.

As providing for the minimum of risk, and as a matter of preference, disposal of a tank effluent by sub-irrigation beneath a cultivated area by means of agricultural pipes or rubble drains is to be recommended. In the Fifth Report of the Royal Commission on Sewage Disposal, it was laid down that "the sewage issuing from septic tanks is bacteriologically almost as impure as that entering tanks," and the discharge of tank effluents into open street channels or gutters and into natural water-courses should, I think, not be permitted.

Following on similar lines of development as those which large public biological installations have undergone, for private tanks we have the two distinct and separate parts, viz.—a sedimentation tank where the liquefaction of solid matter is effected, and filter-beds. In biological methods, the decomposition or conversion of organic matter in sewage is usually divided into the two stages of putrefaction and oxidation. Bacteria are the main active agents in both stages, but in the former they thrive in the absence of air, while in the latter they require oxygen. Temperature, probably owing to the biological nature of the processes, has considerable influence on the workings of septic tanks, and the extent to which this factor operates has been shown by Fuller in an experimental tank at Worcester, U.S.A.(2) "Liquefying" chambers or tanks in their simplest form are just water-tight compartments capable of retaining sewage for a certain period, and provided with inlet and outlet pipes whose openings are submerged below the surface of the contained sewage. The Royal Commission on Sewage Disposal, 1908, affirmed "that the rate of flow through a septic tank is consequently a matter in which the needs of each place require to be taken into account, but from general experience and consideration, we think it may be safely said that at few places should the sewage remain more than twenty-four hours or less than twelve hours in a septic tank." The experiments at Leeds (3) in 1905, showed the best results with a twenty-four hours' flow. This rate has generally been adopted; but with private tanks the sewage reaches the chamber in the freshest possible state, and on this account one would be justified in regarding the capacity for twenty-four hours flow as a minimum.

The amount of sewage entering a private tank may, at any time, be relatively large compared to the total capacity, and this would be a further reason for the twenty-four hour minimum flow.

The use of scum boards, baffle walls, or grilles in tanks has been urged by some observers, and in my experience they have some special application, in small private installations, for equalising flow and minimising possible displacement of scum and deposited matter.

The introduction of a quantity of blue metal or like material of 2½-inch gauge, at and around the opening of the effluent pipe from the tank is advantageous for small installations, and tends to prevent the washing out of solid matter. Owing to the proximity of dwellings, covers for the tanks of private installations are necessary, though, of course, they are not essential for working.

The volume of the daily flow of sewage in different towns varies greatly, and the allowance of 30 gallons per head was suggested by Barwise (4) as a dry weather flow. For private septic tanks intended to meet all ordinary domestic wastes, this allowance appears from observation to be excessive. For these circumstances an allowance of 20 gallons per head per diem is a fair one in my experience, and a tank constructed on that rate will ensure, under all ordinary conditions, a retention of the sewage for a period of at least twenty-four hours, and probably longer.

Like the large installations, contact-beds and percolating or trickling filter-beds have both been employed for small private installations for the purpose of oxidizing and nitrifying the liquefied sewage.

In small installations, the satisfactory and effective distribution of liquefied sewage over filter-beds is a matter of some difficulty.

For contact-beds some automatic movable apparatus is necessary, and in the absence of careful and regular supervision, as has been found in practice, proves more or less a failure. The irregularity and intermittence of the flow of sewage from a single dwelling, as well as in many cases the small amount of total daily flow, militate against the success of contact-beds.

For percolating or trickling filter-beds, distribution of sewage by means of movable and fixed distributors is available. Here, again, the movable variety requires constant supervision, which in the case of private tanks is often lacking and failure results.

Fixed distributors, whether consisting of iron or earthenware with perforations, or pipes provided with holes, or circular metal "spreaders," have in my experience proved most effective and serviceable for small private tanks, being less liable to get out of order, and requiring a minimum amount of attention.

In the metropolitan districts of Sydney, the earliest installations constructed on private premises were chiefly provided with contact-beds. Within the past few years, however, trickling or percolating filters have more or less wholly displaced the contact variety, on account of their simpler and cheaper construction and easier management.

This change has the further advantage, for observers generally are now of opinion that the percolating filter-bed is more effective (5) than the contact. "Through the agency of the percolating filter a nearer approach to securing a rapid means of purification has been made." The chief engineering factors in regard to percolating filters are as pointed out by Herring :—(1) Area of bacterial surface of stones in bed; (2) amount of oxygen available for oxidation of organic matter; and (3) the time of exposure. Free passage of air into the interstices of a percolating filter is a necessary condition for its effective action, and to this end it may be simply a heap of stones without supporting walls, or if supported by walls, they should be liberally supplied with openings for the free passage of air.

The quantity of sewage that can be disposed of on percolating filters varies according to different observers. Barwise has suggested that for filter-beds of 5 feet deep a quantity of sewage equal to 250 gallons per diem per square yard(6) of filtering surface, is a suitable allowance. Generally, the opinion is now held that for properly constructed percolating filters dealing with ordinary domestic sewage, 200 gallons per diem per square yard may be taken as a fair average rate of filtration for permanent work."(7)

From the experiments of Reid carried out at Hanley, it appears that the depth of percolating filter-beds is controlled on the one hand by the strength of the sewage to be treated, and on the other by the size of the filtering material. Practically, he says " that a depth of 4 feet should be fixed as a minimum even for a fine filter," and this has been the standard generally required in the metropolitan districts of Sydney for private installations.

For filtering material, blue metal or any hard non-corroding stone may be used, and naturally, the rate of flow through a percolating filter-bed depends, in some measure, on the size of the particles constituting it. However, almost identical chemical results were obtained at Hanley by Reid with filtering material varying in size from ¾ inch to 1½ inches. These sizes constitute very useful limits for all practical purposes, and have generally been recommended in the metropolis. Where filtering material of size larger than 1½ inches is used, very perfect and even distribution of the effluent becomes imperative.

The possibility of smell sometimes arising from filter-beds is known, and for this reason it is found that persons anxious to employ biological methods of sewage disposal, only wish to construct the closed chamber in which liquefaction of sewage proceeds. The chemical changes that take place in biological installations do not, as a rule, interest the householder, and it is the question of smell which looms largest, if not solely in his ideas. Where a liquefying chamber only is made use of, it would be essential to have a large area of ground, so that the liquefied sewage could be conveyed well away from the dwelling, and an ample portion of land made available for its disposal. In urban areas, as a rule, such large areas are not usually available, and it becomes necessary to make use of the complete installation of liquefying tank and filter-bed. The exposure and agitation that occur during the filtration of liquefied sewage sometimes lead to the liberation of offensive odours due to volatile sulphur and other complex products developed in the decomposition of organic matter.

The escape of these volatile products is more or less unavoidable, without the construction of the filter-bed is such as to largely sacrifice its powers for oxidation and nitrification. Hoods or covers, provided with shafts for ventilation, to enclose the surface of the filter and the distributor, are useful to control the escape of these products. In considering the use of a septic tank on private premises, the possibility of the liberation of offensive volatile products must be considered carefully, both in relation to the house to be served and also to adjacent dwellings and public ways. However, though certainly objectionable, there is little likelihood that the small quantities liable to occasionally arise from well-constructed and managed installations would be productive of any harm.

From observation, during several years, of the large number of private septic tanks existing in the metropolitan districts of Sydney, it has been found that nuisances arising therefrom are comparatively few in number. In the majority of cases it has arisen from the disposal of the effluent, either through neglect to keep soil sufficiently cultivated, or on account of the area provided for disposal proving inadequate or being overtaxed. Where nuisance has arisen from disposal of the effluent, in practically all cases the soil has been markedly clayey, or there has been much outcropping rock. Where such conditions exist, the area for disposal of a tank effluent must be a very liberal one. Where distributors have been of the movable automatic variety, there has frequently been difficulty either through neglect or ignorance of the principles of action, or both. Discharge of storm-water into tanks through ignorance has been responsible for nuisance in one or two instances. Neglect to carry effluents to below low-water mark has also led to nuisance on foreshores. Other causes for complaint have arisen through defective construction, as a result of obvious carelessness or scamping of work by persons engaged in the erection of private tanks. A point of some practical importance to be noted is, that where premises provided with septic tanks for the treatment of wastes are let to tenants, the necessary oversight and attention are invariably lacking, and nuisance has repeatedly resulted under these circumstances. While nuisances have arisen in connection with tanks constructed since the by-laws governing them were introduced, it must, however, be stated that they have formed but a very small fraction compared to those which have occurred in connection with installations erected prior to their introduction.

So far as the experience of the use of private septic tanks in the metropolitan districts of Sydney goes, it must, I think, be admitted that they have proved to be a most useful and convenient method of disposal for household wastes. Constructed and used under proper supervision, they are found to have little, if any, risk. From the standpoints of sanitation and public health they are a decided advance on conservancy or dry methods, and their extensive adoption in unsewered areas has, in all probability, tended considerably to minimise the dangers associated with the storage and disposal of human and domestic wastes.

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THE UNEXPLAINED RESIDUE IN TYPHOID PREVALENCE.

By JAMES JAMIESON, M.D., C.M. (Glas.).
Health Officer, Melbourne City.

THE great reduction in the mortality from typhoid fever in recent times is one of the triumphs of modern scientific hygiene. The extent of that improvement, and the rapidity with which it may be effected, are strikingly brought out in the sanitary history of Melbourne. It remained an unsewered city till it had a population of about half a million, scattered over an area of 250 square miles. A pan system of night-soil collection, largely of a crude sort, was everywhere in use; and from an early period in the history of the city typhoid was very prevalent and fatal. Complaints and protests had often been made, but no kind of concerted action was taken till 1888, when the Intercolonial Medical Congress met. Attention could not fail to be drawn to the insanitary condition of the city, and the Congress adopted a series of resolutions, pointing out in strong terms the crying need of a proper underground system of drainage, with, as its most important accompaniment, abolition of the pan system. The Government of the day was roused to action, and the urgency of the condition was most strongly announced by the occurrence, in 1889, of a typhoid rate of mortality at the alarming figure of about 124 per 100,000 of population. A Metropolitan Board of Works was constituted, and an eminent English engineer was invited to visit Melbourne and report as to the best scheme to be adopted. His report was received in 1890, and in its main features accepted, with some alterations, which involved a narrowing of the area to be first dealt with. The work of constructing the main drains and outfalls was soon entered on, but the first house connection was effected only in 1897. The course of events, in respect of typhoid mortality, since 1888-1889, is shown on the accompanying chart. The first and most notable thing shown is the marked reduction of mortality to about eighty-three in 1890, and to thirty-five in 1891. Similar reductions had happened before, and are quite usual after severe outbreaks of most epidemic diseases; though, in this instance, the decline was greater and more rapid than had before been noted. At any rate, whatever its cause, it could not be ascribed to any considerable improvement in the drainage, and for a few years, up to 1898, there was, if anything, an average increased fatality. Only in 1900 was something like equilibrium reached, with a rate of only 10 per 100,000, much less than any previously observed. That improvement in the drainage, steadily effected, was the most important factor must, I think, be regarded as certain. That much, or any of it, was due to improvement in the water supply can hardly be assumed, as that supply was good when the mortality was at its height, and was not appreciably better in the period of decline. And as to the water supply, it has also to be said that I do not recollect any instance of contaminated water having caused an outbreak of typhoid in Melbourne or suburbs.

Following the indications of the chart, we have this further point to note—that, since 1900, there has been a close approach to uniformity in the curve, the mortality having fluctuated in the last ten years only to the extent of about 3 per 100,000—that is, between 8 and 11. And with this we have got to the point of considering what are the causes or conditions which have led to this persistent occurrence, through a considerable number of years, of even a moderate number of cases.

Water supply has already been referred to as probably almost blameless.

Imperfect or deficient drainage may still, to some extent, serve as a cause, though in an ever-decreasing way. The work of making house connections goes steadily on, and up to the end of June, 1911, no fewer than 112,271 such connections had been completed. There are still considerable areas, in outlying parts of the metropolis, which have not yet reaped full benefit from the sewerage system, and we cannot expect to get the best possible results till the drainage of the whole area is completed.

Contaminated milk is still in some degree a source of danger, and only last year an outbreak was clearly traced to milk derived from a farm at which a typhoid carrier had been at work.

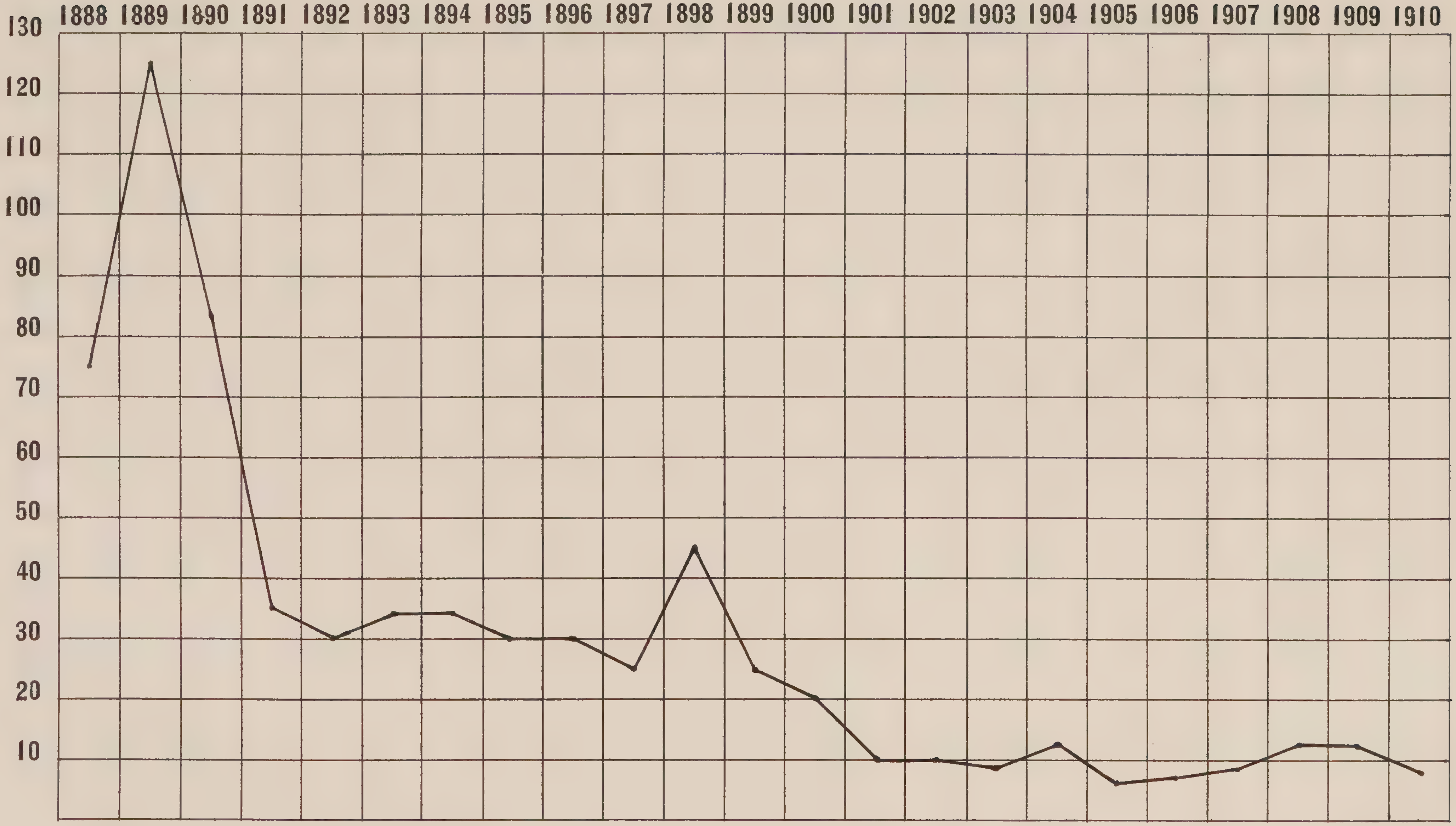
Not so very long ago health authorities had the comfortable feeling that attention to drainage, and to water and milk supply, almost covered their duties in the matter of typhoid prevention. But other and subtler causes and conditions have been shown to be more or less in operation, though mostly in some relation to the great causes just referred to. It may be said, in fact, that almost every case of typhoid is the result of contamination of some article of food or drink, and it is of the utmost importance that this should ever be kept in mind.

Emanations or effluvia from drains or other contaminated surfaces were once accepted as the probable or possible cause of typhoid. This opinion, though now practically abandoned, was, when commonly accepted, one of the chief obstacles to the progress of inquiry into causation. When contaminated water and milk had been excluded, this cause was easy of acceptance, and that its real occurrence, and its mode of action where doubtful or obscure, did not interfere with its persistent acceptance as a real cause.

Direct infection or contagion must still be recognised among modes of transmission. The danger is chiefly from the handling of soiled articles of clothing or bedding. From the relatively high frequency of typhoid among hospital nurses and laundresses we derive at least strong probability of the reality of this cause. Many house epidemics also get their best explanation by the spread of infection from one case to another, whatever be the exact way in which the infecting matter enters the body, whether by inhalation of dried particles or grosser contamination of food.

Flies may, with a probability almost amounting to certainty, be regarded as carriers of typhoid infection. That they carry on and may deposit from their feet many kinds of foulness is certainty. Still it has not been demonstrated, so far as I am aware, that such deposition of typhoid bacilli has actually occurred. Two points, in connection with this possible source of infection, have to be noted. Flies to do this specific mischief must have specific infecting bacilli

Mortality from Typhoid per 100,000 of Population. Melbourne, 1888-1910.



to pick up, and they can hardly do much mischief, even then, if they cannot get access to food. We come back, therefore, to the old point. The more complete and perfect a drainage system is, the less in proportion will be the risk of infection from flies.

Shell fish have been proved to act as agents of typhoid infection, by acting as carriers of the bacilli. At first oysters were alone chiefly implicated, but now mussels are regarded with as much probability as carriers in a similar way. The danger can hardly arise, unless in the case of shell fish growing in the estuary of a river on which stands an undrained or badly-drained town, with consequent gross contamination of the stream. This condition certainly does not apply to Melbourne.

Fried fish, which would ordinarily be regarded as little liable to suspicion, have quite recently been shown, with more than probability, to have caused a whole series of localised outbreaks of typhoid, chiefly in poor districts in London. And this was not owing, as might easily have been believed, to accidental contamination after cooking. An inquiry by Dr. Hamer, one of the Medical Officers under the London County Council, showed that this simple and easy explanation could not be upheld. It was shown that the contamination had been before cooking, and was not produced in course of conveyance to the fish shop, or by the stock of any one wholesale dealer. It had to be assumed that the fish had during life taken up infecting material, and had been imperfectly cleaned and gutted. The general probability of this was strengthened by the fact that the fish were of small sorts, living in shallow waters, where they would be most likely to feed on material carried down by tidal rivers. When the inquiry was made it was, of course, too late for actual proof of specific contamination to be obtained. But the evidence obtained by Dr. Hamer provided ground of inference so strong as almost to amount to demonstration.

A comparatively recent mode of diffusion of infection, often very obscure and difficult to deal with, is by the agency of the *typhoid carrier.* That patients may be, to all outward appearance, fully convalescent and yet continue to throw off typhoid bacilli in urine and fæces for an indefinite period of weeks or months has been clearly established, and the special difficulty in dealing with these cases lies in the fact that they may lead only to isolated instances of infection, and so may escape discovery. It is only, in fact, in the case of an outbreak among the customers of a dairy that attention is plainly drawn to the operation of this cause, as happened in Melbourne about two years ago, and in another instance which I narrated at the Hobart Session of the Congress.

Complete protection from the *carrier* can hardly be got in any easier way than by keeping all known cases of tyhpoid under observation, for bacteriological test as to the freedom from bacilli in the discharges. This involves a large and difficult question for local authorities to deal with, possibly requiring special legislation. The one comforting consideration is that, as the general prevalence diminishes, the likelihood of carriers being met with becomes proportionately less.

And, indeed, when the matter is summed up, we seem to be practically brought back to efficiency of drainage and guarding of food supply. In fact if we brought the matter to an ultimate issue it would be, in the vast proportion of cases, a question of food contamination. Flies, dust, the carrier, would be harmless, or comparatively so, if supplies of food and drink could be adequately guarded. For, in the last resort, we have to recognise that, to the best of present knowledge, the bacilli of typhoid become active agents of infection only by way of ingestion of food or drink. Floating particles in the air may, in a sense, be inhaled into the mouth or throat, but unless swallowed, would probably be harmless; and it is doubtful whether injury would be inflicted by their entrance into the air passages.

It is at the beginning of an outbreak that there is the best chance of successful investigation into the cause of the first scattered cases; and in particular it is at that time that the inquirer may most easily get on the track of the secret carrier. Otherwise minute investigation of the sources of food supply is, and must be, the chief resource of the sanitary officer.

THE SPREAD OF BOVINE TUBERCULOSIS.

By CUTHBERT HALL, M.D., C.M. (Syd.).

EVER since the famous pronouncement of Koch that, in his opinion, bovine tubercle bacilli were incapable of producing the lesions commonly found in the human subject, observation and research have been directed to this question, and the contrary may now be said to have been established, viz., that lesions in man are to be found caused by a bacillus indistinguishable from the bovine. The latest and most authoritative statement, the final report of the Royal Commission on Tuberculosis[1] informs us that "man must therefore be added to the list of animals notably susceptible to bovine tubercle bacilli." The Commission is also of opinion that a considerable amount of the tuberculosis of childhood is to be ascribed to infection conveyed by the milk of the cow.

Of equal importance are the results of the very able study by Professor Delépine as to the influences determining the prevalence of bovine tubercular mastitis.[2] His careful observations have convinced him of the unity of the two forms, and that the milk of cows is the most important factor in the infection of man by bovine bacilli. But he found that in practically all cases tuberculous mastitis occurred late in the disease in cows, for in only one case in 100 did he fail to discover lesions elsewhere when it occurred in the udder, while in three cases the lesions elsewhere were limited. In another series of 400 cows only four were free from other lesions, so that primary tuberculosis of the cow's udder is rare. Again, and this is most important, he found that tubercular mastitis was not common in young cows, whereas old cows in an advanced state of tuberculosis, and emitting discharges loaded with tubercle bacilli, are the chief factors in distribution. As long as there is no ulceration bacilli are retained. Delépine watched one case for many months. During six months there were cough and expectoration; then for four months the milk from two teats was loaded with tubercle bacilli; then that of a third was similarly affected. For the last month the animal suffered from profuse diarrhœa, the fæces being loaded with bacilli, and being dropped in every direction, and swished by the tail over its back, udder, and everything in proximity. The discharge from this cow was dried and kept in a sealed tube, and after nineteen months was found to be still virulent. Hence he concludes finally that bovines with ulcerative lesions, especially of the respiratory organs, alimentary canal, udder, and genito-urinary organs are the chief factor in the prevalence of bovine tuberculosis. Delépine's observations were, of course, the result of work on dairy cows, which were kept more or less in a state of confinement on small farms, and housed and hand-fed or stall-fed, some herds not being depastured on natural grasses at all. Dr. J. Burton Cleland has recently published some important observations on the occurrence of bovine tuberculosis in cattle derived from the extensive stations of Western Australia, and not stalled at all. From these it appears that the disease is fairly prevalent in some districts, and hardly at all in others. Surprise is expressed that cattle living in the open air, with plenty of fresh air and sunshine, are so liable to the disease. But I would remind you that everything points to the fact that cattle are particularly susceptible to the attack of the bovine tubercle bacilli, and that, being gregarious, every chance is given for the spread of the disease. Moreover, the good effects of fresh air and sunshine are discounted by the effects of exposure to all the changes of the weather, heat and cold, drought and shortness of feed and water, such as the stall-fed cow, often housed and rugged at night, has not to endure.

Cattle in large runs, notwithstanding that they may have miles of country to roam over, still maintain their gregarious habit, and especially at night they will camp together in a sheltered spot. As they gather in the same place night after night, this becomes thickly littered with dung, and as they do not bother to choose a clean place to lie on, it will be seen how exposed they are to infection from an animal in an advanced state of the disease. This danger is enormously increased when the dung is dried and pulverised by the trampling of many hoofs and the infected dust inhaled. Probably this is the most frequent means of infection in cattle living in wide, open spaces. But not only may this dust be inhaled, but it must also be deposited on the herbage, and thus be ingested with the food of the cattle. In a country subject to long periods of drought like Australia infected dust is liable to remain on herbage much longer than in countries where rain is frequent, and the dust consequently washed off. Probably infection would take place more frequently were it not for the bactericidal action of the sun's rays. However, germ-laden dust getting on the rough and hairy underside of the leaves of many grasses and herbage would adhere thereto and escape the sun's rays. In very severe drought, too, the cattle are compelled to eat very close, and even to tear out the rhizomes of many grasses, and so must take in as well a certain amount of dust, which may contain bacilli from infected discharges. Cattle have a particular antipathy to eating the grass which springs up round the deposits of their dung, no matter how fresh and succulent and tempting it may be, and they will maintain this aversion for months, so this must afford them some protection. The danger of infection of the pastures has received but scant notice from writers. But it must be remembered that when cattle are turned into a fresh paddock, especially if the grass be tall and succulent, they do not eat it right down at once, but wander about, first browsing off the tops and seeds, and later, when these are exhausted, they come back and feed it off closer and closer to the ground. Thus the pastures must be fouled greatly by the bronchial and other secretions of a beast in an advanced state of tuberculosis, and such animals, I have noticed, drop mucus and saliva and spume freely, especially when rushed about by men on horseback and by dogs Not only so, but such animals frequently, when being rounded up, drop to rise no more. and their flesh and organs are torn and scattered about by dingoes and carrion birds, and so must spread infection around.

We must now consider the influence that the ruminant habit of cattle has upon the spread of the bacilli. If a cow is excreting tubercle bacilli in the bronchial mucus, this is necessarily mainly swallowed, and passes into the first division of the stomach, and mixes with the food, awaiting the "chew of the cud." From the paunch it passes into the reticulum, whence it is regurgitated to the mouth, well masticated, and, when swallowed, passes into the third division of the stomach or psalterium, and passes thence into the fourth compartment, or abomasum. This secretes the gastric juice and has an acid reaction. The stay of the food in this, not being prolonged, its antiseptic action is relatively short, while the reaction of all the rest of the stomach, being alkaline, the bacilli are totally unaffected, and in the case of a healthy cow swallowing or inhaling bacilli, these are in an active condition, and would be able to gain an entrance through an abrasion of the mucous membrane of mouth, gullet, or proximal portion of stomach. Bacilli, when swallowed, are intimately mixed with the food, and when the cud is chewed have a further opportunity of getting lodged in any abrasions, as well as between the teeth, to say nothing of the harsh, raspy papillæ of the tongue. Now a horse seizes herbage mainly by means of his prehensile upper lip, and then bites it off; a cow seizes it by curling her tongue round it and biting it off. The latter thus, if she is excreting bacilli from the lung, and many of these are embedded in the papillæ of the tongue, is more likely to leave some on the herbage in a fresh moist medium to infect another cow who subsequently eats that herbage. Again, whilst a horse's upper lip is covered with hair and dry, that of a cow is smooth and moist, and she is constantly running her tongue over it and protruding the tip into the nostrils, and so is likely to leave bacilli on the nose to also come in contact with the pastures, and leave infection there.

Having then considered how contamination of the pastures can occur, we can readily see how a beast may become infected by them, namely, by feeding off it subsequently and swallowing some of the moist secretions of the previous animal. Moreover, this herbage may be covered with dust from infected dung, which may be swallowed with the grass, or this dust may adhere to the moist nose, and be subsequently licked off and swallowed.

Dr. Cleland has already drawn attention to the water supply as a likely source of contamination and infection. As cattle generally go in a mob twice a day, about 7 a.m. and 4 p.m., to have their drink, and often wash their mouths out to clear them of any stray pieces of grass adhering to teeth and tongue, it can be readily seen that here is a very potent source of infection. This is increased by the manure that is frequently dropped into or about their drinking places, and if not already in the water is washed in by subsequent rains or a rise of the stream or dam. In addition, a weak beast often becomes bogged and dies of weakness and starvation, and the decomposing body, if the organs are loaded with the bacilli, must infect the water.

Although unanimity prevails as to the milk being the source of conveyance of bovine tuberculosis to man, close inquiry does not seem to have been instituted as to how the bacilli gain access to the cow's udder. Human tuberculous mammitis is very rare, and even in the cow it is not of frequent occurrence as compared with the disease in other organs, and it occurs usually only when the disease is advanced in the other organs. One pathway of infection is probably through the blood-stream, the bacilli being conveyed from the other diseased parts. But the most likely mode of infection of the mamma in the cow may be taken to be from contact with infected discharges, and especially the fæces. The teats of the cow are very liable to cracks and abrasions, both when the calf is running at foot and also when she is being milked. If she lies down in infected discharges, or if she gets them on the tail and swishes them about, they are very likely to be lodged in these abrasions, and set up the disease. This is especially likely to happen if there is diarrhœa, and the fæces are liquid.

Attention does not seem to have been drawn to the importance of one habit that cattle possess, viz., that of licking themselves and one another. In this respect they resemble the cat tribe, and have the same harsh, raspy papillæ on the tongue, though the toilet of the latter is much more elaborate. Felines lick their young, but adult felines do not lick one another. Almost the first thing a cow does to her new-born offspring is to lick it all over; hence, if she be tuberculous, she may infect it from birth, even though the disease be latent for years afterwards. The calf of such a cow, too, is sure to inherit a disposition to the disease. Not merely that, but the calf—if the mother is passing bacilli in the fæces and other secretions—is very likely to receive infection from dried excrements on teats and udder, or from these being swished on to it by the tail. A cow will not merely lick every accessible portion of itself, but a friendly mate will often assist and complete the operation. The latter is thus very likely to incur infection from excretions dried on the coat. This licking of their bodies leads sometimes to a very bad habit, namely, that of reaching round and sucking their own milk, a fault not at all uncommon amongst cattle. In a tubercular cow this is probably one of the means of spreading the disease to the udder. Last, but not of least importance, is the fact that before sexual congress the cow and the bull frequently lick one another over very extensively, the latter licking the ano-genital region freely, and thus, if the cow is excreting tubercle bacilli, he is almost certain to be infected, and, later on, when he is in an advanced stage of the disease, he might infect a whole herd.

What practical recommendations may now be made as a result of this study?

(a) The recommendation of Professor Delépine should be invariably carried out, that all cattle with advanced tubercular disease should be destroyed, as they are generally the aged, and, for dairy purposes and breeding, are past the stage of usefulness. Dairy cattle, too, should not be kept more than five or six years, as after that their milk-yield greatly diminishes. I understand it is the rule to inspect all dairy herds in New South Wales when indications of tuberculosis are watched for, and when manifest the beast is destroyed. If suspected, the beast is tested with tuberculin, and if a satisfactory positive reaction is obtained the animal is destroyed. This may hold the disease in check, but it will take more than this to reduce it to a very low ratio, or to vanishing point. Is it not possible that similar inspections be made of the stock on our great cattle stations, so as to destroy all animals with manifest, or even suspected disease? If this were done it would greatly diminish the necessity for inspection and destruction of cattle in advanced states of the disease at our abattoirs.

(b) Though the universal application of the tuberculin test, and isolation or destruction of all positively reacting, may not be considered practicable, yet, from what has been shown of the danger of bulls being a source of the communication of the disease, it would be a step in the right direction if it were made compulsory that all bulls, whether kept for dairy or beef cattle, should be regularly tested, and all reacting positively destroyed. A great step would thus be made in the way of breeding a stock resistant to tubercle, and even by thus working from the male side in a few generations the diminution of the disease would be enormous. The cost of testing bulls ought not to be prohibitive.

(c) Further, as the tendency is to breed more and more from prize cattle and their progeny—and thus, if these are tubercular in a latent form, we are very likely to increase the incidence of the disease by breeding from susceptible stock—it ought to be made a rule with all agricultural societies that no prize be given to any animal that cannot show a clean bill of health—*i.e.*, that cannot present a certificate showing a negative tuberculin test within the previous twelve months. Such a matter would be well worth the cost.

(d) No general examination of the milk for tubercle bacilli is at present made. While this is not necessary, perhaps, still we must remember that bacilli are present in the milk, not merely from mammary disease, but also from fæcal and other discharges. Many diphtheria epidemics have been caused by a dairy employee being a carrier, and probably infecting the milk by spitting on the hands when starting milking, and then dipping them in the milk to keep them moist. It should be compulsory in all dairies that a clean vessel of water be kept, and each cow's teats and udder be washed carefully before milking, and that the milker on no account dip his hands in the milk. Fresh water should be used for each cow, and then the danger from infected manure and discharges getting in the milk will be greatly lessened. Periodical examinations of milk for added water are made, and punishment meted out to offenders. Would it not be as well that similar examinations be made for tubercle bacilli, as a man selling milk with these in is an infinitely greater danger to the community than the man who sells cow's milk diluted to such an extent as to bring it nearer the composition of human milk?

As to the avoidance of contamination of the pastures and water supply, I am afraid little can be done. In the case of the latter, troughs can be kept clean, and dams can be kept clear of manure, and any diseased beasts bogged in the dam or dying in close proximity should be removed and burnt.

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THE MEDICAL INSPECTION OF IMMIGRANTS TO AUSTRALIA.

By A. Wallace Weihen, M.A., M.D., B.S. (Melb.).

At the outset, I should like to clear the ground by stating that, as an Australian, with the interests of my country at heart, I yield to no one in my appreciation of the supreme importance of an active policy of immigration.

In these days of eugenics we must recognise that, apart from education, any attempts to improve race-stocks are limited to two main directions :—

First—Segregation within the limits of a country.

Second—Segregation by refusing entrance to undesirables from without.

It is a truism that a new nation has its whole character and its whole future determined by its first settlers. When subsequent immigration takes place on a large scale in proportion to the total population, fundamental changes in a nation's institutions and ideals are equally to be expected.

We have a unique opportunity to try the effect of hybridising race-stocks on a large scale, and it is a curious thing, but nevertheless, I think, a fact, that while we recognise the value of artificial selection in nearly every department of life, yet we are loth to abandon, with regard to immigrants, the *laissez faire* attitude which I propose to show *does* exist.

"Natural selection" cannot be trusted, of itself, to bring about the best results. "Survival of the fittest" means that those survive who are the fittest for survival, but not necessarily fittest for any other purpose; mere reproductive power is not a test of qualitative value. Nature, who has infinite time to work in, and who can conduct her experiments on a vast scale, can afford to be careless of the single life. We cannot, but we can hasten the production of a finer type.

The case which first directed my attention to the fact that Australia was not making the best deal came under my notice when acting as House Surgeon to an eye hospital in London. One of the staff asked me to give the name of an eye specialist in a certain Australian town, to a patient who was emigrating there to work as a domestic servant. Being interested, I examined the girl, and her condition was such that I took notes on the spot, with the object of sending them out to one of our weekly papers. These are the facts :—

> I.F., aged 21. Seen 2/7/1909. Under treatment for her eyes since 1905.
>
> Right eye had a divergent squint. Vision less than 6/60. Practically blind.
>
> Left eye : Vision 6/36.
>
> Both eyes had nebulous corneæ, due to interstitial keratitis, and very extensive disseminated choroiditis.

Her teeth were classical congenital syphilitic. She informed me that her passage was arranged; she was leaving that week; that the Emigration Officer (who knew of her eye condition, by the way) had told her she would not be examined again, if she was passed in England. To give point to the significance of this case, let me quote Sarah B. Cooper's address to the National Conference of Charities and Correction : "Remember that from a single neglected child in a wealthy country in the State of New York there has come a notorious stock of criminals, vagabonds, and paupers. No less than 1,200 persons have been traced as the lineage of six children, born of this woman."

This case requires no further remarks from me, but an adequate comment on it is furnished by an article in one of the morning papers of the city to which that girl was coming, which appeared on September 13th of last year. It is headed "Domestics—How the Girls are Selected —A Searching Enquiry."

"Complaints are sometimes heard of the quality of the domestic servants who are granted assisted passages, and the Government has been charged for not taking greater care in their selection. This charge is unfounded . . . . . . It certainly, through its agency in London, appears to take all possible care in seeing that the girls who go out are of the best class. In fact, it is not easy to see how the present system could be improved upon. The Medical Officer, appointed by the Board of Trade (not by the Commonwealth, mind you!) to inspect emigrants has repeatedly expressed his high appreciation of the quality of those who obtain assisted passages to this State. "They have always been a most desirable lot," he says; "better people a young country could not, in my opinion, obtain."

I might mention that of 1,700 school children whom I examined in West Australia, the only two cases of typical ricketty teeth I came across were two little girls who had recently come from Sussex.

As Medical Officer of Health in another State I had to examine a batch of immigrants. Having worked among Russians in the East End of London, I had learned the importance attached to an examination of the lids for trachoma in the United States. It greatly surprised these people when I examined their lids, and on questioning some I found that their lids had never been examined before. Now, there is quite enough trachoma here already.

The Superintendent of a city hospital in another State in one week came across a case of morphinism, a case of advanced tuberculosis, and a case of cancer—all moribund, all immigrants, and all arrivals within the previous three months.

Cases of advanced tuberculosis amongst recently arrived immigrants are becoming ominously frequent. A friend of mine recently discovered that his domestic, an immigrant, had been sent out from a consumptive home in England because she was "not doing well"; and another case of advanced laryngeal tuberculosis, seen within three months of arrival here, has come under my notice. I daresay many doctors practising in and around Sydney could tell of similar cases; and it only remains to mention that the number of such cases collecting at our institutions is mounting up, and, to quote Dr. J. W. Pope, who, before the Royal Commission in Perth on Miners' Phthisis, pointed out that "the Government should have power to segregate consumptives. Non-assisted immigrants suffering from tuberculosis sometimes got into the country, not being obliged to undergo medical examination like assisted immigrants."

A great wave of immigration is beginning. At present we are drawing on Great Britain and Ireland chiefly; but, as Mr. John Burns pointed out, we "must not empty the tank," and in any case that source of supply is limited. It behoves the Federal Government, therefore, to bestir itself in order to procure from other countries, which breed good colonists, the stamp of population required.

Precisely! But from what other countries? The experience of America proves that the area from which emigration is taking place has shifted, in recent years, from North and West Europe to East and South Europe.

—"A line drawn from N.E. to S.W., separating Scandinavia, Great Britain, Germany, and France from Russia, Austria-Hungary, Italy, and Turkey, separates countries not only of distinct races, but also of distinct civilisations; lands where education is universal from lands where illiteracy predominates; an educated, thrifty peasantry from a peasantry scarcely a single generation removed from serfdom. This change should challenge the attention of every citizen. Such a change *has* occurred, and it only needs a comparison of the statistics of immigration for 1892 with those of 1902 and 1906 to grasp its extent."

This is what Prescott Hall writes, speaking of America; but the matter is of equal moment to us. We hear that "the wheat lands in Canada are not cheaper than the wheat lands in New South Wales, and considerably dearer than the wheat country of West Australia." This means one thing—we are getting to the end of the cheap lands of the world. "The inference is clear—each year will see more eyes turned toward the empty fertile lands of the Commonwealth."

They are being turned this way, and precisely from the countries we would expect. Who are the mainstay of the labour of many of our mines—Italians. Zangwill some time ago definitely proposed to the then Premier of West Australia to settle a million Russians there on certain conditions. The cry for "more people" to populate Australia has apparently been caught, not only in England but in Russia. "Fifty-four Russian immigrants arrived last week" and so on. A writer from New York recently offered to send to Victoria any number of Italians, Hungarians, and other specified nationalities at £2 a head.

It is surely obvious that we should be prepared with a rigid system of medical inspection. The present is the time to profit by the experience of Canada and the United States, and to get in early, because even in the case of immigrants from Great Britain it is chiefly the poor who come.

We cannot expect shipping companies or emigration agencies, or countries of departure, to look after our interests too tenderly. Only within the last few years an incident occurred which threw a flood of light on this point, and showed what is still a common point of view, when a magistrate in an English provincial town remitted sentence on a prisoner on condition that he promised to emigrate to Australia.

What are we doing to safeguard ourselves?

1. At port of departure.—There is no routine examination; the Commonwealth requires nothing. Some of the States—*e.g.*, West Australia and New South Wales—require a medical certificate, which is usually a perfunctory and valueless performance. The West Australian Government is now instituting examination by its own men—but this scheme is "in embryo."

The first big fact to note is that non-assisted immigrants are not required to present a certificate at all; assisted immigrants to New South Wales obtain the following certificate, which is presented to the Agent-General, and they are admitted practically on this certificate. I was under the impression that the certificate of any doctor was accepted by the States (with the possible exception of Queensland), but recently an *ex cathedra* statement has been made that "each State has a separate doctor dealing with intending immigrants." This is certainly a move in the right direction, but I understand no special qualifications are insisted on.

This is a copy of the certificate :—

> "I certify that I have this day examined A.B., intending immigrant to N.S.W., and found him in health and of sound constitution. He is not suffering from any mental or bodily defect which, in my opinion, unfits him for (work). Both his hearing and eyesight are good."

There is a note at the bottom : "To avoid needless hardship, the examining physician is requested to satisfy himself that the applicant is in every way a fit subject to pass a thorough medical examination, as applicants are liable to rejection after inspection on the ship" (presumably by the ship's doctor).

This note, in view of the fact that the above certificate is practically our only line of defence, is rather a näive request to the doctor to do his best for us, as we cannot help ourselves.

On Arrival.—All people entering the Commonwealth are medically examined at first port of entry. Any person found to be a "prohibited immigrant" is left to the Customs to deal with. The actual inspection is done by the Medical Quarantine Officers. The Customs report to the Department of External Affairs, which deals with such cases principally on the basis of their liability to become a charge on public funds.

This inspection has its sanction in clauses b, c, d, f, of section 3 of the Immigration Restriction Act, 1901-1910, defining prohibited immigrants. If prohibited immigrants are discovered on board, permission to land is refused, and the matter lies between the company and the passenger. If a case has been once allowed to land, the State has to pay—there is no hold on the shipping company.

Last, there is the significant fact that no statistics are available as to the medical causes of rejection of immigrants, and probably not more than fifty have been deported in the last two years.

That is the system, and I venture to make the following comments :—

(1) In justice to the Immigration Department, one has to note that undesirables are, as a rule, non-assisted immigrants.

(2) The present inspection and certificate are not nearly explicit enough. Their general nature, though apparently intended to give them a drag-net character, makes it easy for a slipshod or interested examiner to do as he likes. They will weed out an applicant with heart disease, say, who if he came out here would not hand on his defect to his progeny, but I think it would be comparatively easy for a case of congenital disease (such as I gave earlier in this paper), or one with Hutchinson's teeth, to get through and become the progenitor of children of a type which goes largely to populate our asylums and goals. This to my mind is a matter of infinitely greater importance.

Surely it should be possible for our legislators to consult the medical profession, which is slowly winning recognition as the custodian of the public health, on a matter of such vital interest as this, with a view to drawing up a scheme of standard requirements to which every immigrant, assisted or non-assisted, should conform; such conformity to be certified to by medical men, who should be whole-time officers, retained by the Commonwealth. These men should be men of experience, not only in general practice, but also in the special departments in which particularly one gets evidence of inherited and transmissible disease.

(3) Clause b of the Act—with regard to persons likely to become further charges—though at first sight salutary and effective, is largely inoperative, because it is most difficult to show that an immigrant becoming a public charge does so from causes arising prior to landing. Of all the aliens reported as public charges in the United States in 1904, only 19 per cent. were actually deported. This proves the regrettable fact that, even under the U.S.A. system, the medical certificate is not the controlling factor that it ought to be.

(4) I am convinced that there is only one adequate way of protecting Australia, and that is to have the medical examination here on the lines followed in America. Of course the point will be at once raised that we are much further away, and that therefore such a method is unworkable. That is a matter of practical politics which I do not think I am called on to discuss. Another point of objection is that of hardship to the immigrants. This I hope to show later can be dismissed as negligible.

Now let us see how Canada and the United States handle this matter. I quote first from the Dominion Year Book for 1908, Report of Chief Medical Officer, Department of the Interior :—" The work of this service includes the inspection of all steerage and second-class passengers landing at Atlantic and Pacific ports, or coming *via* U.S. ports."

" With a view to obtaining accurate knowledge as to the character of the immigrants, special inquiries have been undertaken to determine the extent of diseases in immigrants, expressly as regards insanity, feeble-mindedness, tuberculosis, and trachoma. Obviously, except in the case of inmates of public institutions, the information gleaned lacks the definiteness of tabulated statistics; but as immigrants, who get sick, if of limited means, soon must seek charity, we may fairly assume that relatively few cases escape notice."

In addition there is the trans-Atlantic Medical Inspection, and, in the case of immigrants from Great Britain receiving any official assistance, the following particulars must be supplied on a regular printed form :—

(1) General appearance—strong? vigorous? delicate? ruddy? pale?

(2) Apparent height, apparent weight.

(3) Has applicant any obvious physical defect or malformation? Give details.

(4) Is he feeble-minded, idiotic, epileptic?

(5) Is he insane or has he had an attack of insanity within five years?

(6) Is he deaf and dumb, deaf, dumb, blind?

(7) Is he infirm? If so, give details and state if he is going with his family or to family already in Canada.

(8) Is he affected with a loathsome disease or with a disease which is contagious?

(9) Is he a pauper, destitute, professional beggar, or likely to become a public charge in Canada?

This is signed by applicant and by the responsible officer of the Society assisting.

The following are the details of the Medical Certificate in connection with assisted immigrants required by the Canadian Government :—

(1) Is physical history of applicant's family good? If not, state defects.

(2) Do the sounds of the chest, as ascertained by percussion and auscultation, indicate a perfectly healthy condition of the lungs?

(3) Is there any predisposition to tubercular disease of the lungs, hereditary or otherwise?

(4) Have any relatives died of phthisis, bronchitis or other lung disease?

(5) Is the constitution of the heart healthy?

(6) Is there any tendency to epilepsy or fits of any kind?

(7) Has applicant ever had rheumatic fever?

(8) Is applicant ruptured?

(9) Has applicant ever had trachoma or suffered from disease of the eye? Is applicant's sight good?

(10) State the condition of the teeth.

(11) Is applicant suffering from eczema or any other skin disease?

(12) Has applicant undergone an operation? If so, what?

(13) When was applicant last vaccinated?

(14) Has applicant any organic defect or bodily infirmity?

(15) Is applicant strong physically? mentally?

(16) Has applicant or any relation at any time been the inmate of a lunatic asylum? If so, when?

(17) Has he had any serious disease or injury? If so, of what nature?

(18) Is any such affliction likely to recur ?

I hereby certify that the abovenamed person has been examined by me, and that I am of the opinion that he is of sound constitution, in robust health, mentally fit, and a suitable person for emigration to Canada,

(Signed) A.B.

Subsequently each immigrant must pass the inspection of the Port Officer appointed by the Board of Trade, as well as the shore Medical Officer of the steamship line, and finally the Medical Officer of the vessel before being taken on board. In addition to this, the Medical Officer and Master of the vessel must certify to a certain bill of health before the immigrants can be removed from the vessel.

When, in addition to these various measures taken to sift out undesirables, an examination of each person is made by one, and at the large seaports by two, Medical Officers, followed by a thorough civil inquiry into the age, occupation, destination, and financial standing, and an estimate of the moral qualities and likelihood to succeed in Canada and become good citizens, it would appear that little more could be done to prevent the ingress of improper persons into the country.

In the criticisms made on the medical inspection of immigrants one question is always asked, viz., "Have Canadian officials inspected at foreign ports all immigrants before they take their passage?" Remembering the number of seaports at which immigrants coming from different countries embark, knowing how all those in any way assisted are required to present certificates as to physical and mental health, and realising that a very large proportion of immigrants go to some one of the thousands of booking agents in these several countries, buy their tickets inland and arrive at the steamer only a few hours before sailing, it will be apparent that no effective scheme can be devised for dealing with these several classes of cases, other than requiring intending passengers to be present in the seaport towns several days before sailing, to undergo a rigid examination before being allowed to go on board.

What seems, however, quite practical is a further detailed and thorough examination during the voyage and a daily observation by the ship's doctor of every person on board, subject to inspection. He is required at present to certify in the manner indicated (bill of health), and if the company can be compelled to enforce, and such officer can be stimulated to make, such examination an exact and serious matter, by filling in blank forms with particulars of the examination of each immigrant, every practical requirement would be fulfilled, for the attention of the doctor inspecting at the port of landing would be called to any case about whom any remarks were made, and a further special investigation of such could be made after the general inspection was over. It is not unreasonable to expect that such systematic examination would be gladly agreed to by every steamship company, as it would lessen the risk of bearing expense of deportation, and delay at the seaports of landing, as the result of the detailed examination of each immigrant, which many critics demand.

Next—Statement of Immigrants detained and debarred in Canada in 1908:—

Total Immigrants	262,469
Detained	4,638
Debarred	1,172

Looking up the Commonwealth returns in 1910 under the Immigration Restriction Act, we find :—

Total persons admitted...	94,543
Persons refused admission	42
Persons refused admission under clauses b, c, d, section 3...	5

Either we are getting the healthiest people in the world, or we are not weeding out undesirables.

Turning now to the Canadian statistics of causes for which immigrants were detained in 1908, one notes first the striking fact that for contagious diseases only 22 were detained. Under the section (eye diseases) one finds—

	Detained.	Debarred.	In Hospital.
Trachoma	1,073	635	16
Conjunctivitis	1,810	3	39

So more than half of the cases detained were due to eye conditions, and more than one-third of cases rejected were due to eye conditions.

In view of the facts that in our system the eyes are not rigidly inspected, and that these Canadian immigrants had, previously to setting out, passed through a fairly severe overhauling, I find these results somewhat startling. We have already more than enough trachoma in Australia, and we are likely to get more through an influx of Russian and other aliens. When House Surgeon at Moorfields, I was interested in the question of the sources of trachoma, and analysed all the cases of trachoma, attending there consecutively throughout one week. I found that 76 per cent. were aliens, and of these aliens practically 95 were Russians.

The United States system of examination is modelled on the same rigorous lines. Lack of time prevents me from entering into details, and from showing how conclusively the history of American immigration proves the imperative necessity for such care, and from bringing under your notice the perfection of the arrangements for inspection at Ellis Island.

Our quarantine system guards us pretty effectively against the introduction and spread of acute infectious disease in immigrants, but the greatest risk to which this community is exposed is by the influx of large numbers of persons of poor physique, who would lower the general vigour of our population. The experience of other countries has been that such persons are those who are most likely to become a burden on the community. The importance of a proper physical test has been emphasised over and over again. Thus the Commissioner for New York in 1903 in his report estimated that of the million immigrants for that year 200,000 were an injury instead of a benefit, and he advocated a physical examination, and the exclusion of those who failed to fulfil its requirements, on certification to that effect by the U.S. Marine Hospital Surgeons.

In 1904, the Chief Surgeon at Boston reported: "Superiority in quality is no longer characteristic of the immigration at this port. To some extent this low physical standard is the result of the extension of the business of the British lines into Continental territory, but there seem also to be a decided increase in the physically and mentally defective from Great Britain and Ireland as well."

Again let me quote: "Of all causes for rejection outside of dangerous, loathsome, or mental disease, that of poor physique should receive the most weight, for, by admitting such aliens, not only do we increase the number of public charges by their inability to gain their bread through their physical inaptitude and their low resistance to disease, but we admit, likewise, progenitors to this country whose offspring will reproduce, often in an exaggerated degree, the physical degeneracy of their parents."

It may be objected that the more rigid the restrictions the less will be the total immigration, and the greater the proportion of persons debarred. The experience of the United States emphatically negatives these two points.

Intending immigrants learn the standard of exclusion as well as the methods of evasion, so that the proportion who take the chance of failing is very small; still, deportation is a hardship and should be avoided. The United States have rejected, as ineffective, all proposals for relying on inspection abroad, because they do not "strike at the root of the evil, which is the business energy of the steamship companies (and agents) in soliciting immigration, and their business caution in requiring doubtful immigrants to give bonds in advance to cover the cost of carrying them back. It is not the exclusion law that causes hardship, but the steamship companies (and agents) that connive at evasion of that law. Their surgeons and officials know the law and its standards as thoroughly as the immigration officials, and it only needs an adequate motive to make them co-operators with the Immigration Department instead of evaders of the law.

That adequate motive the law of 1903 provided in the form of a fine for every alien debarred and a responsibility on the shipping company offending to carry the alien back at its own expense. It is now suggested, I believe, to make such responsibility operative within five years after the landing of an immigrant. In Canada we know that a company offending must deport within two years if called upon to do so.

Here in Australia, so I understand, when once the immigrant is landed, the shipping company is relieved of responsibility, *ipso facto*, and further, in the case of such as have been deported, the expense has been shared between the shipping companies and the State. Comment on this as a business proposition is hardly necessary! Now assisted immigrants cannot be deported, I believe.

Actual exclusion is only a small part of the total process of rejecting the undesirable. The theory of the law of exclusion is that the shipping companies will, through their agents and for their own protection, refuse to sell tickets to immigrants who will be unable to pass the test. In our case it is not necessary for a shipping company to worry a single jot about a non-assisted immigrant.

What attitude we may expect shipping companies and agents to adopt if we do not safeguard ourselves, will be seen from the following facts.

1st. That in the case of doubtful immigrants to the United States, shipping companies have demanded double passage money, with the object of making a profit on carrying the immigrant back in case he is rejected. This is proved by the action of the Italian Government in passing a law giving an immigrant a right to recover money retained illegally by a shipping company in this way.

2nd. A circular issued by one of the great transportation companies says (I quote the report for 1900 of Commissioner-General for Immigration) that a diseased person who claims to be able to prove American citizenship on arrival will be accepted for passage if he deposit with the company 150 dollars, such amount to be forfeited to the company if he is deported on arrival. The same circular thoughtfully provides "that a person so afflicted shall agree to be isolated from the other passengers during the voyage." In effect the circular points out an easy means of getting in, to such diseased immigrant, if he is willing to guarantee the company against loss, and make a false declaration of American citizenship, and there are plenty of fraudulent naturalisation papers in use even now.

3rd. Coaching immigrants in the methods of evading the law and passing the examinations is extensively carried on, and I know, for a fact, that it is quite a common practice for immigrants to the United States who have "doubtful" eyelids to use adrenalin just before landing to help them past an examination for trachoma.

Now, I think it will be conceded that there are many undesirables who are ready to come to Australia, and it is equally possible that there are many who are willing, for a consideration, to help them to do so. Other countries are rejecting them—they must emigrate. Emigration to Australia, therefore, if we continue on our present lines, will be for them the path of least resistance, and we will inevitably get these rejects.

While we are prepared to extend a warm welcome to all who are in earnest to make Australia their home and add to her strength and wealth, yet we have an equal right to refuse to allow ANY to make our country the scene, either of their ineffectiveness, their follies, or their crimes.

THE INTERNATIONAL NOSOLOGICAL CLASSIFICATION AND ACCURATE CERTIFICATION OF CAUSES OF DEATH.

By G. H. Knibbs, C.M.G., F.S.S., F.R.A.S., Federal Statist.

SYNOPSIS.

1. Adoption of International classification in Australia.
2. Revision of nomenclature.
3. Translation of revised nomenclature.
4. Defects in certification.
5. Remedy.
6. Conclusion.

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1. Adoption of International Classification in Australia.

Under the authority of the Minister for Home Affairs, I published in February, 1907, a translation of the "Nomenclature of Diseases and Causes of Death," revised and adopted by the International Commission in 1900, and published by that Commission in French. This translation was made available by an issue (gratis) throughout Australia, to all medical men who desired a copy. By this means the preparation of statistics under this nomenclature was much facilitated. This translation referred to attracted attention in America, and the fact of the adoption of the international nomenclature by the Commonwealth Bureau of Census and Statistics was enthusiastically appreciated.

2. Revision of Nomenclature.

The question of co-operating in the revision of the nomenclature was taken up in Melbourne by Dr. Maudsley, then honorary secretary of the Eighth Australasian Medical Congress, and by Dr. W. P. Norris, then President of the Board of Health, Melbourne. A small committee was appointed in 1908, consisting of Dr. W. P. Norris, Dr. J. Jamieson, Dr. Springthorpe, and myself, as Federal Statistician, and asked to submit, in the light of Australasian experience, a scheme of revision, in order that it might be formally brought before the International Revision Committee meeting in Paris on the 1st to 3rd July, 1909. Ordinarily the revision would have been in 1910, but to permit of the 1910 United States census tabulations being developed in conformity therewith, this revision was advanced one year on the original intention. The small committee referred to met, and a number of suggestions as to the additions to the list of those causes of death that were not mentioned in the international classification, but nevertheless certified in Australia, was prepared. This list included everything that, so far as was known, ought to be considered in the international revision. The suggestions of the committee were adopted by the Medical Congress, and in due course were forwarded with some notes by myself, on 8th December, to Dr. Jacques Bertillon, of Paris, who was specially charged with the care of these matters, and whose labours have associated his name with the nomenclature.

In the meantime, a communication was made through diplomatic channels to the Governor-General of the Commonwealth, asking that Australia should be represented on the committee of revision referred to above, and I was appointed to attend, which I did. After three days' consideration, during which the majority of the recommendations of the Australasian Medical Congress were adopted, the whole matter was remitted to Dr. Bertillon to be put into suitable form. The delegates agreed to recommend to their respective Governments the adoption from 1st January, 1910, of this revised nomenclature, so as to secure uniformity and comparability of statistics of causes of death, the countries represented being Australia, Austria, Belgium, Brazil, Bulgaria, China, Colombia, Costa Rica, France, Greece, Italy, Mexico, Monaco, Montenegro, Netherlands, Rumania, Russia, Spain, Switzerland, and the United States.

3. Translation of Revised Nomenclature.

While in Paris I conferred with the United States delegates, and agreed upon a common translation of the nomenclature, so that the American and Australian editions should be identical. They are, in consequence, almost *verbatim et literatim* the same.

Commonwealth vital statistics have, of course, been compiled as agreed under this new classification, so that they are comparable with the vital statistics of the other countries using it. As arranged, they will be classified from 1st January, 1911, under the revised nomenclature.

In this connection it may be mentioned that New Zealand has now adopted the new classification, as from 1st January, 1910, and from the 1st January, 1911, it has been adopted for the whole of the United Kingdom. I understand that it is intended in future that the mother country shall be officially represented on the International Revision Committee.

4. Defects in Certification.

Unfortunately, the adoption of a more or less satisfactory nomenclature is only one step in the direction of getting good statistics. The compilation of the whole in the one Bureau (*i.e.*, the Commonwealth Bureau of Census and Statistics) has the advantage of securing uniformity in the interpretation of the certificates. But the results necessarily depend on the accuracy of the certificates themselves; behind this the tabulation cannot go.

At the present time, since certificates of cause of death are open to the members of the family to whom they are delivered, it is not possible to secure accuracy in all cases. The certifier in many instances will not, for example, distress the family by stating the real cause (*e.g.*, in cases of venereal disease).

I have discussed the question of the extent and characteristics of misstatement of cause of death with Dr. W. P. Norris, with the Hon. Dr. C. K. Mackellar, and with Dr. J. H. L. Cumpston, at present Acting Director of Quarantine for Australia, and to the lastnamed I mainly owe the indication of defects likely to occur when the certification of death is open to inspection by the family of the deceased, instead of being secret. The social objection to open certification is a strong one, and we cannot hope to receive accurate certificates or to obtain a proper knowledge of causes of death while the existing system continues. This is evident if we but consider the causes likely to be miscertified. The following list, in which the numbers are those of the last international nomenclature, gives the necessary indication:—

List.—37 Syphilis; 38 gonococcus infection; 56 alcoholism; 64 alcoholic cerebral congestion and apoplexy; 113 alcoholic hepatitis and alcoholic cirrhosis of the liver; 59 morphinism, cocainism, nicotinism, chronic ether intoxication; 62 locomotor ataxia; 67 general paralysis of the insane; 74 onanism, &c.; 127 non-venereal diseases; 140 puerperal insanity; 125 urinary abscess, &c. This list is illustrative, not exhaustive. It has been pointed out that with open certification the certificate would state local cause without specifying general conditions—for example, "syphilitic stricture of the œsophagus" should be classified under 37, but would probably be certified merely as "stricture of the œsophagus," and thus would be erroneously classified under 101; "gonorrhœal pyosalpinx*" would probably be certified simply as pyosalpinx,* the alcoholic origin of cirrhosis of the liver would probably be suppressed. Neurasthenia would probably be certified instead of the drug habit, leading to classification under 74, instead of under 59.

5. Remedy.

The preceding instances will sufficiently indicate the nature of the grave defects of the existing system of open certification. At the last Congress (see Appendix I, Transactions, vol. I, p. 17) it was resolved: "That, in the opinion of the Congress, a form of death certificate, which would facilitate uniform and specific certification of the causes of death, is most desirable, and any such certificate should be forwarded direct to the local registrar of deaths, who should issue a burial certificate"; and in Appendix II (*loc. cit.*) a form of "death certificate" was suggested, which would appear to leave nothing to be desired. But accuracy of certification has undoubtedly not yet been made effective. Nevertheless, the whole matter may be very simply dealt with. All that is necessary is that in all cases the death certificate given to the family in which the death occurs should be in a sealed envelope addressed to the Registrar, and the Registrar should be required to transmit these monthly to the central authority for tabulation, the envelopes and forms being supplied by the Central Government undertaking the tabulation, and being the property of the Government supplying them. The Registrar could require of the family that on the registration of death the envelope should be intact, and it would be quite sufficient if the family were suitably informed orally of the cause of death, this merely for their own comfort, but not for any legal reason.

It is, of course, deplorable that no inconsiderable portion of the statistics of causes of death is invalidated by intentionally erroneous certification. Statistical tabulation is a considerable expense, but unless it represents the facts as well as they are known, it is questionable whether this expense is justifiably undertaken. The statistician rightly limits himself to the declarations of the medical certificates; his interpretations of these certificates are restricted merely to the bringing of the assigned causes of death under the nosological nomenclature adopted for statistical purposes. Notwithstanding the knowledge that systematically erroneous certification exists, he would not be justified in tabulating according to conjectural variations of the frequency of alleged causes of death. It is clear, therefore, that some scheme of proper certification should be adopted without further delay. Other countries have been able to secure it, why not Australia? Switzerland, for example, a country of similar population to Australia, has organised a system of correct certification of causes of death, and there is no reason or hindrance whatever, except public apathy, why we should not follow its lead. We should long ago have actualised a method of correctly ascertaining the frequency with which, in any particular way, disease and death strike at our community. The solution of the difficulty is quite simple if it be readily attempted.

In the country referred to, viz., Switzerland, the certification of the cause of death is made direct to the Federal Statistical Bureau, where the whole tabulation is undertaken, and probably in no country is the work more thoroughly, expeditiously, and satisfactorily done.

The form of the certificate approved by the eighth session of the Australasian Congress needs no material amendment for the purpose in view; all that is necessary is to add an identifying name, address, and date. The address is to facilitate localisation. It will then stand as follows:—

DEATH CERTIFICATE.

The cause of death of (*name in full*) of (*here write address*) on (*here write date*) was as hereunder written:—

Cause of Death.	Duration of each Cause.†			
	Years.	Calendar Months.	Days.	Hours.
(1) Disease causing death*				
(2) Immediate cause of death				
(3) Complications and contributory causes, if any				

Note.—Neither (2) nor (3) need be filled in if doubt exists as to the item.

Confirmed by }
Not confirmed by } *Post-mortem.*

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* In Death by Violence state—(2) Mode of injury, accidental, suicidal, or homicidal; (2) Nature of the injury and immediate cause of death; (3) Contributory causes or conditions.

† The duration of each form of disease or symptom is reckoned from its commencement until death occurs.

6. Conclusion.

The conclusion of the whole matter is that it is only through confidential certification on a suitable form to the Registrar of Deaths by the medical attendant in each case, and by tabulation under a unified scheme, that statistics of causes of deaths can be rendered of high value. Even if this involved some slight amendments of State laws, such amendments would be more than justified, and they could be easily effected. From the standpoint that an intelligent study of social phenomena is properly to be expected in the public interest, these reforms are necessary. Statistics, in which the essential element is partially or wholly suppressed, is of inferior value, since by transferring cases from the cause properly assignable to another cause to which the case ought not to be assigned, it vitiates in each instance two tabulations.

THE SCOPE AND ORGANISATION OF A SCHOOL MEDICAL SERVICE.

By Dr. Mary Booth.

A striking development in preventive medicine in this century has been the recognition of the valuable service it could render to the cause of education. Medical inspection of schools and school children has become a definite State function in most countries with modern systems of education. The schemes vary greatly in the reasons for their employment, in comprehensiveness, and in method. They have thrown into relief the conception that the physical wellbeing of the child not only as an individual but as a potential parent and citizen is of primary importance to the State.

Broadly speaking, we may say the scope of medical inspection is the study of the school child as a growing organism, and of the influences, hereditary and environmental, which promote or hinder the full development of his mental, moral, and physical powers. The school surveys to date have amassed already a vast amount of such material, and there is evidence in many directions of a desire among the more thoughtful school hygienists for a critical study of the work done, so that the real problems of social hygiene may be more definitely stated.

The organisation of a school medical service must be, at this stage, the formulating of methods by which these problems can be elucidated, and the reforms indicated carried out. A very considerable part of such an organisation will be the establishment of working relations with existing health and sociological agencies, so that there shall be a reinforcement of effort instead of an overlapping or neutralisation.

The first point for critical study is in regard to the child. How often and to what extent must a child be examined for an effective minimum? How shall the results be classified? How far shall treatment, the necessary corollary of medical inspection, be compulsory and free?

In regard to school environment. How and by whom shall the school be controlled in the matter of construction, furniture, and use, the school programme of work, and in regard to communicable diseases? Further, the equally or even more potent environment of home—how shall its influence be estimated and modified where necessary? For both the school and home, the Public Health Authority, and for the home the social worker, can give invaluable help.

Again, in regard to the education of the child in hygiene, the school medical service must see that provision is made to develop in the child a health conscience—that even such problems, suitably set out and led up to, as an efficient birth-rate, a low infant mortality rate, an efficient citizen soldier, are essential to the public good. Lastly, the larger view must be constantly before the school medical service of watching the trend of the national physique by periodic anthropometric surveys. The medical examination of children, all important as it is, is really a palliative measure. We know, from examinations already made, that a large percentage of defects is preventable. The amassing of statistics of defects, and even of satisfactory treatment, must not be allowed to obscure the necessity for research into causes and radical reforms.

I know nothing that gives a better perspective of the scope of school hygiene than a short survey of its beginnings. On the European Continent, apart from the classic surveys of Cohn and others, it began in France with the duty of keeping school-houses clean. In Sweden, as a means of detecting children unfit for physical training. In Germany, as an inquiry into epidemic eye disease; and later, at Wiesbaden, a complete medical inspection of the child in the modern sense was begun. In America, as a means of detecting and controlling communicable diseases—the first school doctors of Boston were employed to visit the schools in the morning session to exclude any affected child. A start was made with fifty physicians, the city being divided into fifty districts. The method spread rapidly, and in some States to-day embraces some of the most elaborate physical examinations in existence. In England, apart from many instances of private enterprise, the public movement developed directly from special inquiries into the welfare of defective children—for example, Commissions inquiring into the number and condition of the "blind, deaf, and dumb, the crippled, and the mentally defective" leading up to the Royal Commission into Physical Deterioration, with the organisation of Swedish physical training throughout the schools. After a period of partial and permissive legislation, the code of 1908 made it the duty of the Board of Education to examine medically every child entering school, and as often afterwards as they deemed necessary.

In Australia, it is interesting to note, the first systematic medical examination of the child took place as part of the systematic anthropometric survey of school children carried out in Sydney in 1901. There were two main objects in view—(1) to put on record the physical data of the Australian child and the number of the generations his family had been in Australia, so that the development of the Anglo-Celtic race mixture in Australia and the trend of the national physique could be studied; (2), to ascertain the extent to which physical defects existed among school children. In the different Australian States, where systems of medical inspection have been more recently established, we are passing through the experimental stage, but are already realising that we have difficulties and problems peculiar to ourselves, due in part to climate, and in part to the scattered population, and in part to our better average social circumstances. In the objective study of these, especially in comparing the work of the different systems, we should find indication for more profitable use of our energies and economic organisation. To return to the practical considerations. The examination of the individual child. It is probable that the complete examination of every child every year is unnecessary, and that three examinations during the eight years of school life, as proposed in England, will give the best results.

In fixing the age or school period at which the first complete examination should be made, we should take into account Australian conditions. The time fixed in England is at entrance, but we must remember that very many of the Board School children in England come from centres of dense population and faulty home conditions. In Australia, on the whole, poor social condition is rare, and at any rate, the children, who are its victims, stand out in marked contrast to the average. On the other hand, there are a great number of our Australian schools which show such faulty hygiene that the children's health positively deteriorates from attendance at them. I would, therefore, at this stage of school construction, place the first thorough examination at the beginning of the second year of school life—that is, the upper-first class, so that defects due to school conditions, and other defects, shall be detected and provided against. It seems to me that for the young growing organism, the great and unnatural alteration in the mode of life that "going to school" means should be carefully checked by the school authority.

If the school examination were fixed by years of school life, it would include all children retarded from any cause. It would require also that all other children who have suffered from certain specified diseases, and those obviously of poor physique or ill health, shall be specially selected for examination.

Another complete examination should correspond to the early pubertal period—the stage of physiological high pressure. It is a period which has yet to be determined for Australasian children, and which may be found to vary in different latitudes, as, for example, Tasmania and Northern Queensland. The proposed anthropometric survey of Australian children will be very valuable in showing whether there is any deviation from the British standards. The importance of ascertaining the period of rapid growth in boys and girls is obvious when one considers how much our educational system is governed by the demands of public examinations, and how the physical training of the junior and senior cadet, if not apportioned according to the physical facts of his growth, may do him irreparable harm.

Another factor affecting the question of extent and frequency is whether the school to be examined is urban or rural. Urbanisation means ready spread of communicable disease among the children, especially poorer muscular development, and also, it is said, poorer vision. Gulick says, "Man, having harnessed the powers of nature, no longer uses his muscles as heretofore," and the change affects the children: that the other heritage of the children is giving time for play, space for play, tradition for play. It is not, therefore, incumbent on us that the examination of the health of the children in cities shall be more constant than in the country? Here, again, a careful comparative study of results for the children of agricultural and mining districts and the industrial town artisan and professional population would be the best guide to a decision.

A final examination of the child should take place in his last year at school.

I am aware that in fixing these important transition stages of life, viz., "going to school," "puberty," and "leaving school" as the periods which should be selected for examination, one is making what may seem a difficult proposition. But I think a rough correspondence to age and class groups, with certain known exceptions, will be found. To establish such a correspondence is one of the pressing problems of anthropometry and school hygiene for Australia.

The school child cannot be dealt with effectively apart from its parent and teacher. Co-operation of the parent has been gained in Victoria by sending him, prior to the examination, a card with printed questions as to the heredity, the home environment and habits, and the previous history of the child. The parents have responded in probably about 98 per cent. The co-operation of the teacher has been most willingly given in taking part at the time of the examination, and filling in the cards before it. One of the most stimulating experiences of a school medical officer is to find how profoundly the revelations of a medical examination affect the attitude of a teacher to a child.

It is very desirable that the work of medical inspection shall appeal to the general body of the medical profession. It has revealed the existence of a great deal of unhealthy conditions, due to faulty hygiene in infancy and early childhood, which, though not severe, yet depreciate the physical efficiency of the child—for instance, minor manifestations of rickets, dental decay, sequelæ of measles and scarlet fever.

The organisation of a nursing service is essential to the success of medical inspection—not so much for giving assistance to the medical staff at the time of the examination as for "following up" the cases whose parents are too ignorant or too helpless to obtain the treatment suggested for their children. The nurse gets home to the people, and can be a most sympathetic and helpful factor in the lives of these people. She forges the links between the home, the school, and the State. She co-operates with the sanitary authorities and the agencies which are doing invaluable social service. To these social agencies in the future we must look for help in our study of hereditary factors.

In regard to treatment, it is useless to lay down individual schemes. The most satisfactory way of meeting the problem is for the education authorities to approach accredited medical associations and ask for their friendly co-operation in its solution. To arrive at a working scheme will not be an easy matter. But there is no one but the medical profession itself that can deal with it effectively.

The school environment of the child should be such that he is the better physically for having spent some years there. Yet, even in our brief history, we have been able to burden ourselves with architectural structures, which cause actual deformities in and devitalise the children, which we cannot afford to pull down *en masse*, though we are aware of the harm they are doing. We must not repeat this. There should be attached to every Education Department an architect who would specialise in school architecture, and confer with the school medical officer and teacher as to the design and structure and control of the building. The special climatic conditions of Australia should be investigated in regard to school conditions, and on the scientific findings public health legislation for State and non-State schools alike be based. Liberal playing spaces and open-air classes should minimise greatly the chest conditions that answer for so large a mortality in less genial climates.

The sanitary control of the environment should be in the hands of the public health authority, which has a machinery always at work. The present unsatisfactory arrangements which exist in some of the States, whereby the public sanitary authority cannot take action until a technical "nuisance" is created, leads to neglect or a deadlock. It is particularly in regard to procedure in the case of epidemic disease that a clear statement of responsibility is necessary. Even if the Public Health Act does not prescribe the duties in regard to school premises, there should be mutual agreement between the public health and education authority as to notification, quarantine, disinfection, and in the case of diphtheria "carriers," so that at least the child does not suffer.

Again, the prevention of tuberculosis through the agency of the school by the repeated examination of cases known to have been exposed to the infection, is a matter of organisation where tuberculosis is a notifiable disease.

One other condition, the dirt diseases of children, only now beginning to be recognised as serious in their relation to other diseases, may require some compulsory cleansing measures, as have been found necessary abroad. Prosecution of these cases, which are essentially due to house and clothing infection, without the provision for facilities for cleansing, is persecution.

Another function of a school medical service is the detection and classification of the mentally deficient, and of educating the public to the importance of their special care and control. In this work there should be co-ordination with the Department of Lunacy and any private agencies interested in these unfortunates.

A recent development affecting the health of the school-boy and adolescent is the inauguration of the cadet system of the defence scheme. It would be a saving of time, energy, and money if one medical examination served for the cadet and school examination. The question of physical endurance and fatigue and physical training must be ever present in the mind of the school medical officer.

The development, co-ordination, and control of these many and varied activities of a school medical service demand a medical department, which should be attached to the Education Office with considerable freedom of action. The central staff should exercise a general supervision over the assistant staff, should undertake special surveys, and be equipped with necessary clinical room and laboratories for medical or psychological research.

The extension of the work to the scattered country districts presents a problem peculiar to Australia. There, however, the local practitioner may take part; and, with the assistance of the bush nurse giving part of her time, valuable work could be done. The difficulty of extending the work in the less remote and urban centres is one of expense only.

Engagement of the local practitioner as part-time officer, with the co-operation of a district nursing service, will probably be the solution. A promising beginning has been made in Victoria in this direction through co-operation with the Bush Nursing Association. The council of the latter have established nurses in two remote country districts, and arranged with the Education Department for certain school duties. The bush nurse has an unusual opportunity for establishing relations between the school, the home, and the community. She assists the school medical officers when she visits the district. She examines the teeth, hair, throat, skin, and clothing of the children at intervals. She attends to minor ailments, and reports to the parents any cases she considers should have medical advice. She gives simple practical teaching in First Aid and personal and domestic hygiene. A monthly return of work done is sent to the Education Department. The bush nurse, in the one case where definite arrangements have been made, is a part-time school nurse giving the equivalent of a day a week to school work, the Government paying, approximately, one-sixth of her salary. It is recognised that, owing to the nature of her duties as bush nurse, her work in the schools must be somewhat irregular. The school teachers are instructed to meet her convenience as much as possible.

That each State of the Commonwealth has developed independently its own scheme, I have always regarded as a fortunate circumstance. I think the time has come, however, for the medical officers of the different States to meet in conference, so that some minor points, such as uniformity in estimate of physical defects for statistical purposes, might be agreed upon. Another urgent matter is the question of transfer of unhealthy children from one State to another. A child, for instance, suffering from tuberculosis or syphilis should have to present himself to the Education Department of the State he was entering, and come directly under the care of the school medical officer. This should be arranged either through the Education or Public Health Departments, but the school medical officer should be directly cognisant of it. By such a conference we could attract the interest of the general body of the profession to our work, and then help in the solution of its problems.

In matters of even greater importance, as the relative value of certain investigations, we should find much to discuss. Take, for instance, the effect of the humid climatic conditions of the North of Australia and Papua, and its influence, if any, on the physique of the Australian child—the importance of an anthropometric survey as a quantitative test. We should arrive by meeting thus at a full consciousness of the possibilities of school hygiene.

It is on the true conception and co-ordination of the manifold duties of a school medical service that the future of Australia to a great extent depends.

THE SECULAR PROGRESS OF PULMONARY TUBERCULOSIS AND CANCER IN AUSTRALIA FOR THE PAST THIRTY YEARS, THEIR ANNUAL FLUCTUATION, AND THEIR FREQUENCY ACCORDING TO AGE.

By G. H. Knibbs, C.M.G., F.R.A.S., F.S.S., Federal Statistician.

SYNOPSIS.

1. Introduction.
2. The secular fluctuations of pulmonary tuberculosis.
3. The secular fluctuations of cancer.
4. Constancy of rates for total deaths from pulmonary tuberculosis and cancer.
5. Masculinity of death-rates of pulmonary tuberculosis and cancer.
6. Monthly fluctuation in the frequency of death from pulmonary tuberculosis and cancer.
7. Frequency of death from pulmonary tuberculosis and from cancer according to age.
8. Death-rates from pulmonary tuberculosis and cancer in each age-group.
9. Conclusions.

1. Introduction.

Tuberculosis and cancer are diseases which stand in a remarkable statistical position in regard to one another, inasmuch as though, pathologically, apparently without relationship to one another, the sum of the deaths resulting from them in any given year, per million of population, is almost constant. This is the more remarkable since this constancy is collateral with a great diminution of the frequency of deaths from tuberculosis; in other words, the rate of increase in deaths from cancer has been such as to almost exactly balance the rate of diminution in deaths from tuberculosis.

The following indicates the recorded causes of death included under each of the above headings :—

Pulmonary Tuberculosis.—Tubercular broncho-pneumonia, plastic bronchitis, consumption, pulmonary consumption, phthisis, fibroid phthisis and pulmonary phthisis, phthisis, pulmonary and laryngeal, tubercular pleurisy, tubercular pneumonia, tuberculosis unqualified, and tuberculosis under the following heads, viz. :—Acute, capillary, miliary, acute miliary, miliary and laryngeal, miliary and renal, pleural and meningeal, pulmonary, pulmonary and abdominal, pulmonary and arthritic, pulmonary and cerebral, pulmonary and gastric, pulmonary and dermal, pulmonary and intestinal, pulmonary and general, pulmonary and laryngeal, pulmonary and glandular, pulmonary and meningeal, pulmonary and pleural, pulmonary and renal, pulmonary and spinal, pulmonary and sacral.

Cancer, Nos. 39 *to* 45 *inclusive.*—Includes cancer, epithelioma, carcinoma, malignant tumours and diseases, neoplasm, sarcoma, rodent ulcer, and scirrhus.

For the reason indicated above the two diseases have been considered together in this paper. In the results for the Commonwealth, those for Western Australia are not included before 1897, as complete records for earlier years were not available. Nevertheless, the results may be regarded as applicable to the Commonwealth throughout, as the Western Australian experience for the period omitted was of very limited extent.

2. The Secular Fluctuations of Pulmonary Tuberculosis.

In the following table the frequency of death from pulmonary tuberculosis is expressed in deaths per 100,000 of the corresponding sex, and for the entire population. The results are given for each year and the quinquennial means also, inasmuch as the progress is better seen from these means. The great regularity of the figures is worthy of notice.

DEATH-RATES from Pulmonary Tuberculosis in Australia for thirty years per 100,000 of the corresponding sex, or population.

Year.	Deaths per 100,000.			Year.	Deaths per 100,000.		
	Males.	Females.	Persons.		Males.	Females.	Persons.
1881	136	112	125	1896	110	84	98
1882	147	115	132	1897	104	80	93
1883	141	111	127	1898	112	86	100
1884	154	112	135	1899	102	80	91
1885	151	113	134	1900	105	77	92
Mean (1–5)	146	113	131	Mean (6–10)	106	81	95
1886	143	105	126	1901	106	81	94
1887	141	106	125	1902	105	78	92
1888	142	101	123	1903	102	79	91
1889	136	94	117	1904	98	81	90
1890	136	97	118	1905	92	69	81
Mean (6–10)	139	101	121	Mean (1–5)	100	77	89
1891	130	89	111	1906	87	68	78
1892	123	89	108	1907	83	71	77
1893	128	89	110	1908	85	76	80
1894	124	87	106	1909	79	67	73
1895	117	87	102	1910	76	63	68
Mean (1–5)	124	88	107	Mean (6–10)	81	69	75

The above results are shown on Graph No. 1, see curve A thereon.

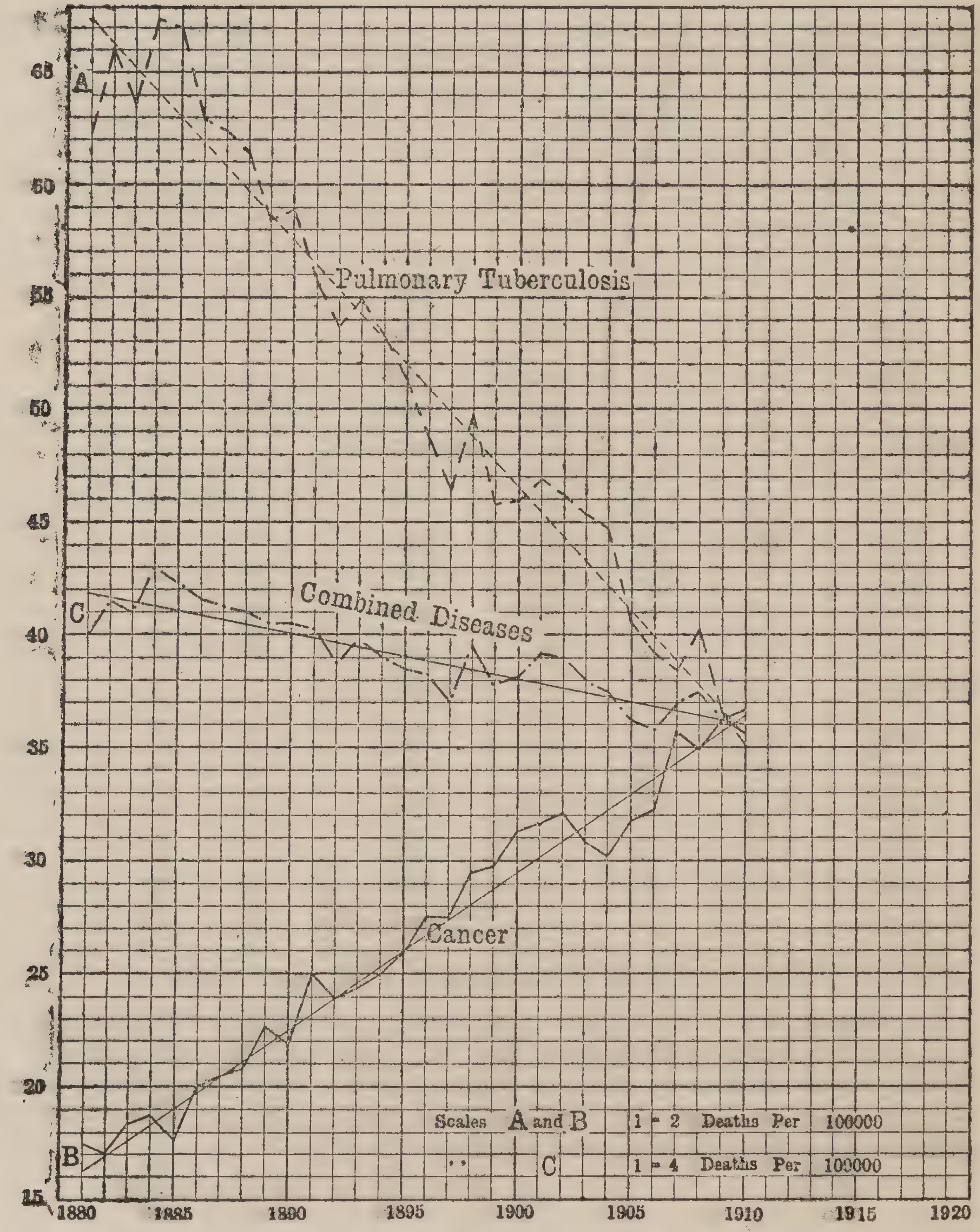
An examination of the diagram illustrating the successive death-rates from tuberculosis, discloses the fact that the general trend is linear, that is, varies uniformly with the lapse of time. Solving by the method of least squares from the quinquennial means we get the following formulæ for males, females, and persons, viz. :—

Males ... ... $D = 157 - 2{\cdot}6\,(T - 1880)$
Females ... ... $D = 115 - 1{\cdot}7\,(T - 1880)$
Persons ... ... $D = 137 - 2{\cdot}2\,(T - 1880)$

in which D is the number of deaths from tuberculosis per 100,000 population of the same sex, or total population—as the case may be—and T is the year for which the mortality is sought. The position of this general trend for persons is shown by a fine broken line on the diagram. The deviations of individual years are seen to be small.

COMMONWEALTH OF AUSTRALIA.—DEATH-RATES FOR PULMONARY TUBERCULOSIS AND CANCER, 1881 TO 1910.

Graph No. 1.



Explanation of Graphs.—The base of each small square represents an interval of one year, while the height in the cases of curves A and B represents one death per 50,000 of population; and in the case of C, one death per 25,000 of population. In the cases of A and B, the base line (15) represents 30 deaths per 100,000; and in the case of C, it represents 60 deaths per 100,000. Thus, for curves A and B, the numbers on the left-hand column have to be doubled; and in the case of curve C, have to be quadrupled to give the number per 100,000. These curves indicate the linearity of trend of the diseases, and the fact that this trend is in opposite directions.

3. The Secular Fluctuations of Cancer.

The frequency of death from cancer is expressed in the following table per 100,000 of the corresponding sex and the entire population. As with pulmonary tuberculosis, not only are the results for cancer given for each year, but also the quinquennial means. The steady frequency of deaths from the latter disease is as remarkable as that observed above for the former.

DEATH-RATES from Cancer in Australia for thirty years per 100,000 of the corresponding sex, or population.

Year.	Deaths per 100,000.			Year.	Deaths per 100,000.		
	Males.	Females.	Persons.		Males.	Females.	Persons.
1881	33	37	35	1896	56	54	55
1882	35	33	34	1897	55	55	55
1883	36	37	37	1898	61	57	58
1884	35	40	37	1899	61	58	60
1885	36	35	35	1900	59	67	63
Mean (1–5)	35	36	36	Mean (6–10)	59	58	58
1886	37	43	40	1901	67	59	63
1887	41	40	40	1902	67	61	64
1888	40	43	42	1903	63	60	62
1889	45	46	45	1904	58	63	60
1890	45	42	44	1905	65	62	64
Mean (6–10)	42	43	42	Mean (1–5)	64	61	63
1891	53	46	50	1906	64	65	64
1892	48	48	48	1907	74	69	71
1893	50	47	49	1908	69	71	70
1894	51	49	50	1909	72	73	72
1895	54	49	52	1910	77	72	73
Mean (1–5)	51	48	50	Mean (6–10)	71	70	70

The tabulated results are shown on Graph No. 1, see curve B thereon.

The general trend is linear as with tuberculosis, and from the quinquennial means we obtain the mortality rates for males, females, and persons :—

Males ... ... $D = 32 + 1{\cdot}4\,(T - 1880)$
Females ... ... $D = 31 + 1{\cdot}4\,(T - 1880)$
Persons ... ... $D = 31 + 1{\cdot}4\,(T - 1880)$

D denoting as before number of deaths from cancer per 100,000 of population of the same sex or total population, and T the corresponding year. Here, again, the deviations are not large, as will be seen by a reference to the diagram for deaths from cancer in successive years; see curve B, on which the straight line shows the general trend.

4. Constancy of Rates for Total Deaths from Pulmonary Tuberculosis and Cancer.

As already mentioned, it is found that the diminution in deaths from tuberculosis is nearly but not quite equalled by the increase in deaths from cancer. The figures are very steady for either sex or both combined, as the following table shows :—

DEATH-RATES from Tuberculosis and Cancer together in Australia from 1881 to 1910, per 100,000 of the corresponding sex, or population.

Year.	Deaths per 100,000.			Year.	Deaths per 100,000.		
	Males.	Females.	Persons.		Males.	Females.	Persons.
1881	169	149	160	1896	166	138	153
1882	182	147	166	1897	159	135	148
1883	177	148	164	1898	172	143	158
1884	189	152	172	1899	163	138	151
1885	187	148	169	1900	164	144	154
Mean (1–5)	181	149	166	Mean (6–10)	165	140	153
1886	181	148	166	1901	173	140	157
1887	182	146	165	1902	172	139	156
1888	182	144	164	1903	165	139	152
1889	181	140	162	1904	156	144	150
1890	181	140	162	1905	156	132	145
Mean (6–10)	181	144	164	Mean (1–5)	164	139	152
1891	174	136	161	1906	151	133	143
1892	171	137	155	1907	157	139	148
1893	178	136	159	1908	153	147	150
1894	175	136	156	1909	151	140	145
1895	171	136	154	1910	153	134	142
Mean (1–5)	176	136	157	Mean (6–10)	152	139	146

These tabulated results are shown on Graph No. 1, Curve C, on which the straight line shows the general trend.

The steady movement of the figures is equally remarkable for either sex, and varies linearly with the lapse of time as expressed by the formulæ :—

Males ... ... $D = 189 - 1{\cdot}2\,(T - 1880)$
Females ... ... $D = 146 - 0{\cdot}3\,(T - 1880)$
Persons ... ... $D = 168 - 0{\cdot}8\,(T - 1880)$

in which as before, D is the sum of the deaths from both diseases in a population of 100,000 of the same sex or both combined, and T is the year for which the mortality is to be found. On the diagram the fine line shows the general trend and discloses how small the deviations are for individual years; see curve C.

These last formulæ, independently determined, are also given as the sum of those previously given in sections 2 and 3. It may be reiterated that the special feature of these two diseases is not only that the trend of the death-rate of each is sensibly linear, but that it is so for each sex as well as for the sexes combined. Consequently, year after year, the improvement in the rate of death from pulmonary tuberculosis is practically nullified by increase in the rate of death from cancer, forasmuch as the decrease and increase respectively in the number of deaths per million (or 100,000) of population are sensibly the same.

The Australian constancy in the sum of deaths from cancer and pulmonary tuberculosis is shown also in the statistics of England and Wales for these diseases. There, also, the death-rate for pulmonary tuberculosis has diminished while the death-rate for cancer has increased. The following table indicates this :—

DEATHS per million from Pulmonary Tuberculosis and Cancer in England and Wales (Males), 1901 to 1909.

Year.	1901.	1902.	1903.	1904.	1905.	1906.	1907.	1908.	1909.
Pulmonary tuberculosis.	1,487	1,456	1,427	1,458	1,347	1,356	1,341	1,310	1,270
Cancer	691	696	732	740	756	794	781	813	826
Both diseases ...	2,178	2,152	2,159	2,198	2,103	2,150	2,122	2,123	2,096

Mean 2,142.

These results show that for the period 1901–9, the diminution in death-rate from pulmonary tuberculosis in England and Wales, and the corresponding increase in death-rate from cancer are sensibly uniform in operation, and that the number of deaths per 1,000,000 may in each case be represented by a linear function. In the case of pulmonary tuberculosis, cancer, and both diseases combined, this function is :—

Pulmonary tuberculosis ... $D = 1{,}282 - 21{\cdot}6\,(T - 1900)$
Cancer ... ... $D = 825 + 13{\cdot}2\,(T - 1900)$
Both diseases combined ... $D = 2{,}107 - 8{\cdot}4\,(T - 1900)$

D denoting the number of deaths per annum per 1,000,000 of population, and T any year in question.

Comparing these with the figures already given for the variation in the case of Australia, we have the following, the English rates being reduced to rates per 100,000 for the sake of comparison, and t denoting the year in question, reckoned from 1900.

	Australia.	England and Wales.
Pulmonary tuberculosis	$137 - 2{\cdot}2t$	$128 - 2{\cdot}16t$
Cancer	$32 + 1{\cdot}4t$	$83 + 1{\cdot}32t$
Both diseases combined	$169 - 0{\cdot}8t$	$211 - 0{\cdot}84t$

In each case the slope of the straight line representing the trend is practically identical for Australia and England and Wales.

5. Masculinity of Death-rates of Tuberculosis and Cancer.

When death-rates for the two sexes, instead of remaining constant, change in any definite way with the lapse of time, the masculinity, or frequency with which deaths of males occur, as compared with deaths of females, will in general also change. When the death-rate for each sex has been deduced, and the law of its progression has been determined, then the masculinity can be deduced for a short range of values.* It is, however, better deduced directly from quinquennial mean values of the masculinity. The values for the masculinity of tuberculosis and cancer are per 1,000 females—

Tuberculosis ... ... $M = 1{,}386 - 5{\cdot}3\,(T - 1880)$
Cancer ... ... $M = 983 + 2{\cdot}2\,(T - 1880)$

and these show the variation of the relative probability of death for the two sexes from either disease. This, however, varies with age, as may be seen from Graphs 3, 4, and 5.

6. Monthly Fluctuation in the Frequency of Death from Pulmonary Tuberculosis and Cancer.

The frequency of death from tuberculosis and cancer varies from month to month. To get really comparable results it is necessary to furnish them for months of equal duration, so corrected also as to correspond to a constant population, since in the case of Australia, the deaths are drawn from a population steadily increasing from January to December. The corrected mean of four years' records, viz., 1907 to 1910, gave the following results, viz. :—

MONTHLY variation of frequency in deaths from Pulmonary Tuberculosis and Cancer, Australia, 1907–1910.

Deaths per month per 10,000,000 of population.

Month.	Jan.	Feb.	Mar.	April.	May.	June.	July.	Aug.	Sept.	Oct.	Nov.	Dec.
Pulmonary tuberculosis.	609	561	584	563	626	618	675	699	671	712	594	601
Cancer ...	612	634	606	607	610	593	568	581	614	584	581	620
Total...	1,221	1,195	1,190	1,170	1,236	1,211	1,243	1,280	1,285	1,296	1,175	1,221

Mean 1,227.

The curves indicating these fluctuations are shown on one of the circular Graphs, viz., No. 2, diagram A.

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* Thus, suppose the death-rate for males be—

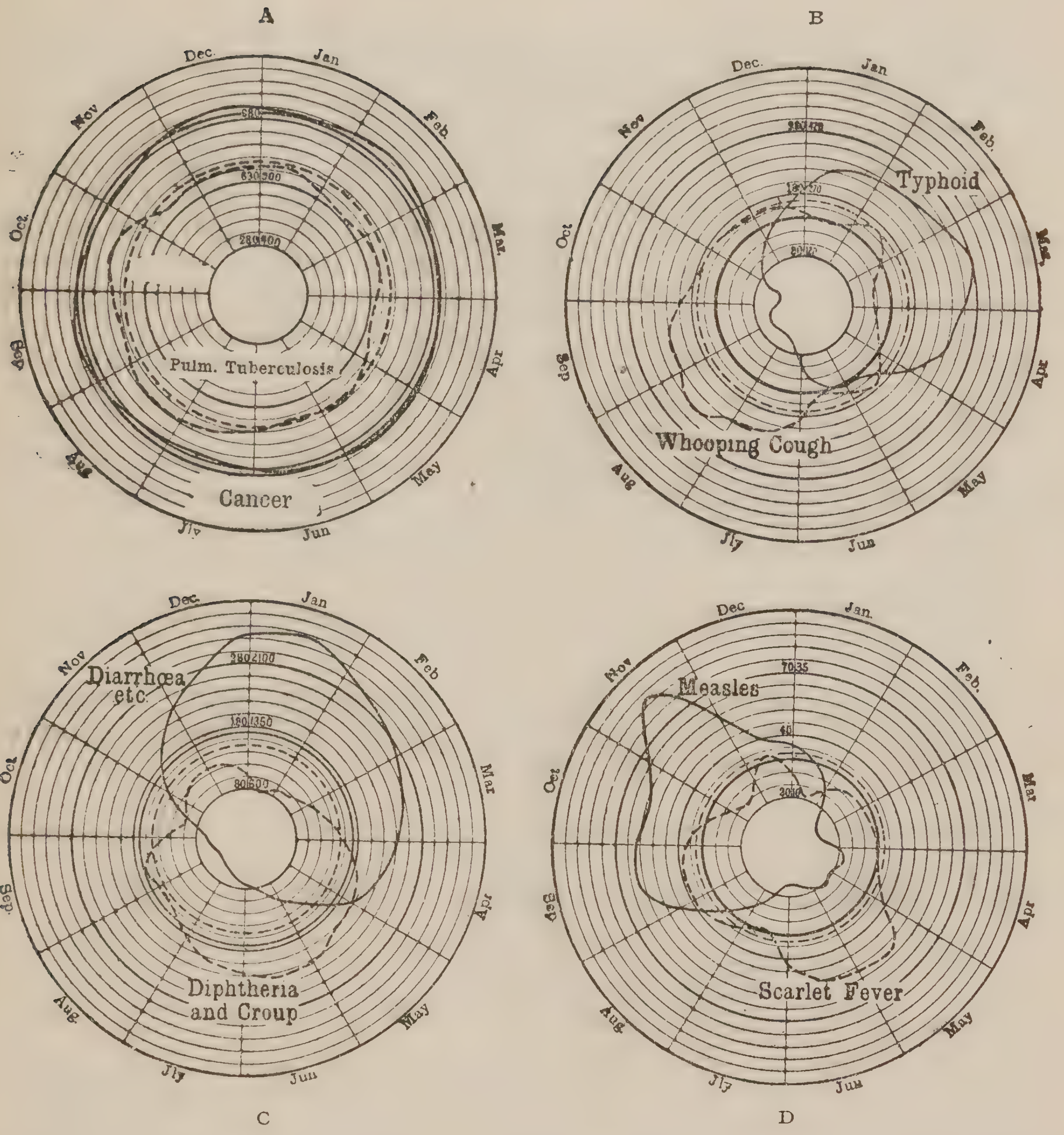
$$D = A\,(1 + at)\,;\ \text{and for females be}\ E = B\,(1 + bt)\,;$$

where A and B, and a and b are constants derived from past results, and t denotes time elapsed from any epoch or date arbitrarily adopted for convenience; then the masculinity is—

$$m = D/E = A\,(1 + at)\,/B\,(1 + bt) = \frac{A}{B}\left\{1 + (a - b)\,t\right\},\ \text{approximately.}$$

COMMONWEALTH OF AUSTRALIA, 1907 TO 1910.

Graph No. 2.



Explanation of Graphs.—The graphs based on the monthly totals of deaths over the period 1907–10 show at any moment the relative frequency of the following diseases, viz., pulmonary tuberculosis and cancer; typhoid and whooping-cough; diarrhœa, dysentery, and enteritis, and diphtheria and croup; and measles and scarlet fever. For each disease a circle is drawn (a continuous or a broken line as the disease is represented by a continuous or broken curve) showing the position corresponding to a uniform distribution throughout the year, and this indicates whether the value at any particular time is above or below the average for the disease. The numbers on the diagram denote the aggregate per month per quadrennium, and the intervals between each small circle represent the following numbers respectively where A denotes Arabic figures and E Egyptian figures.

Cancer	A :	1 interval	represents	70	deaths.
Pulmonary Tuberculosis...	E :	,,	,,	100	,,
Whooping Cough... ...	A :	,,	,,	20	,,
Typhoid	E :	,,	,,	30	,,
Measles	A :	,,	,,	5	,,
Scarlet Fever	E :	,,	,,	2·5	,,
Diphtheria and Croup ...	A :	,,	,,	20	,,
Diarrhœa, &c.	E :	,,	,,	150	,,

7. The Frequency of Death from Pulmonary Tuberculosis and from Cancer, according to Age.

The statistics of the years 1907 to 1910 furnish data for determining, approximately, the frequency in age-groups of deaths from tuberculosis and from cancer. The population has been approximately corrected for the preliminary census count. The results are given for males, for females, and for persons in the table hereinafter. In a previous paper, communicated to the Eighth Session of the Australasian Medical Congress, 1908, Vol. II, pp. 199–200, I gave a diagram (Fig. 4) showing the age-groups and curve based thereon for pulmonary tuberculosis, as deduced from Australian statistics for 1907. I gave also, for comparative purposes, a diagram (Fig. 3) showing results for England and Wales for 1906. The maximum frequency of death for males appeared to occur at age 38½, and for females at age 23½ (*vide* p. 200). The result was not given as here for both sexes together (persons), but was at age 33 to 35 approximately. The more extended results now given would indicate a maximum for both sexes combined at ages 26½ or 27, which is obviously not in very good agreement with the one year's result previously given.

The table hereunder gives the results for males, females, and persons :—

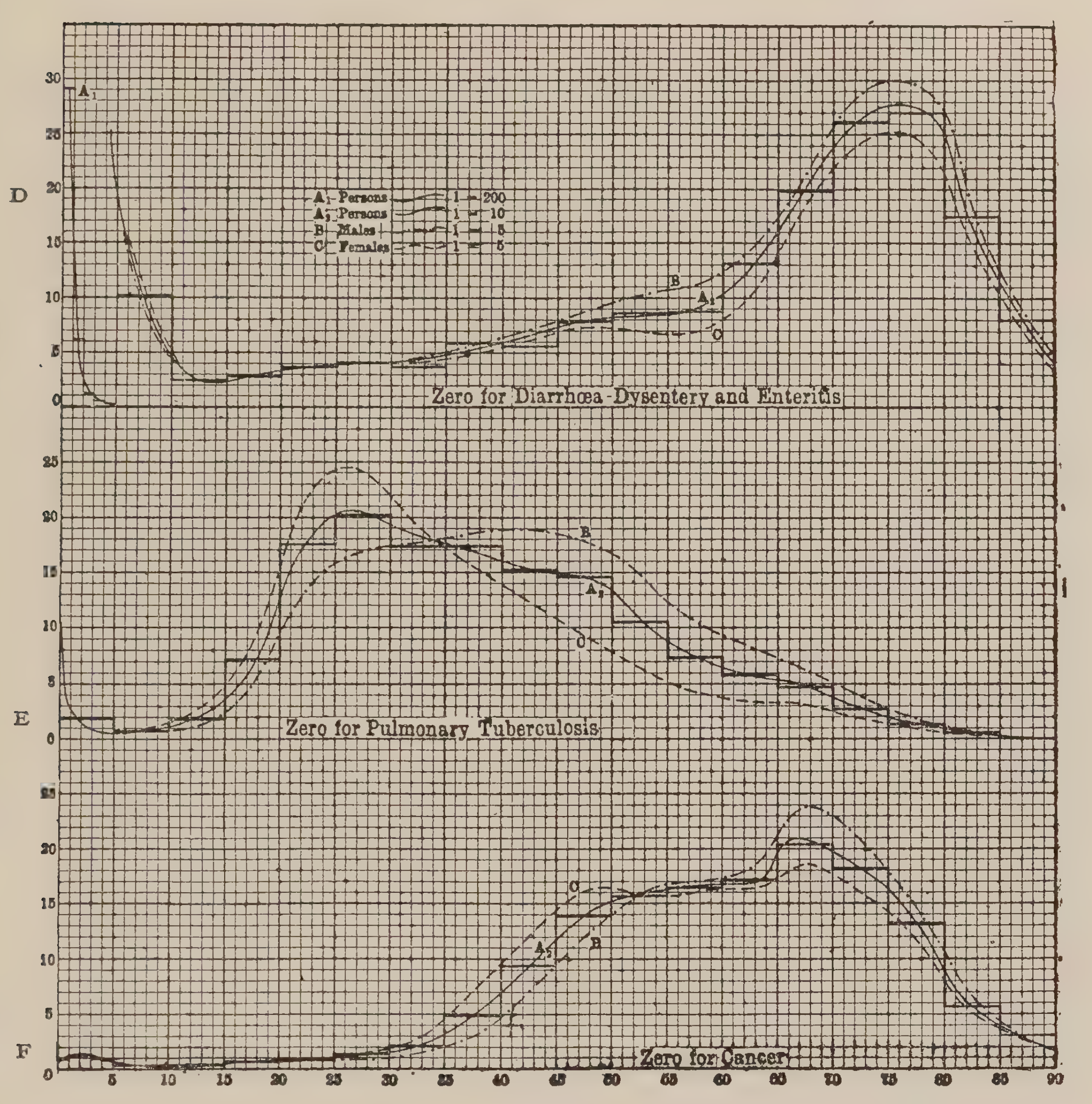
FREQUENCY of Death per annum in Age-groups from Pulmonary Tuberculosis and from Cancer per 10,000,000 of total population per annum, Australia, 1907–1910.

Age-group.	Pulmonary Tuberculosis.			Cancer.			Age-group.	Pulmonary Tuberculosis.			Cancer.		
	M.	F.	P.	M.	F.	P.		M.	F.	P.	M.	F.	P.
0	21	21	42	5	4	9	35–39	495	377	872	83	159	242
1	19	12	31	6	5	11	40–44	472	287	759	189	283	472
2	8	4	12	5	7	12	45–49	496	237	733	312	380	692
3	4	4	8	5	4	9	50–54	389	139	528	409	373	782
4	1	4	5	4	4	8	55–59	268	99	367	442	377	819
5–9	18	20	38	15	6	21	60–64	207	85	292	467	389	856
10–14	31	66	97	8	10	18	65–69	167	74	241	594	429	1,023
15–19	170	195	365	17	13	30	70–74	98	41	139	527	382	909
20–24	363	524	887	23	17	40	75–79	44	21	65	367	288	655
25–29	447	566	1,013	26	36	62	80–84	11	7	18	160	121	281
30–34	447	425	872	37	67	104	85–	11	...	11	84	66	150

M. denotes males, F. females, and P. persons.

COMMONWEALTH OF AUSTRALIA.—DEATHS AT VARIOUS AGES, FOR VARIOUS DISEASES.

Graph No. 3.



Explanation of Graphs.—The curves represent the number of deaths per annum at each age in a total population of 10,000,000, corresponding to the Australian experience for the four years 1907–10, the population of 10,000,000 being assumed to be distributed according to sex and age in the same proportions as the mean Australian population for 1907–10. In each case the continuous line represents persons, the broken line represents females, and the dot and dash line represents males. The base of each small square represents one year of age, while the height represents 200 deaths in a total population of 10,000,000 in the case of curve A 1, ten deaths in the case of curve A 2, and five deaths in the case of curves B and C.

It will be seen that there is a characteristically different incidence of death from pulmonary tuberculosis in the two sexes, as was pointed out in my paper previously referred to (see diagram Trans. Med. Congr., Vol. II, p. 199). The three curves are shown in the diagram, but the values of age-groups are given on the diagram only for Persons. The maximum frequency of death from pulmonary tuberculosis for Males is probably at about age 42,* for Females at about age 26½, and for Persons, as said, at 26·7 years. The diagram referred to, shown above, is the middle one of the three (see Graph No. 3, curves E).

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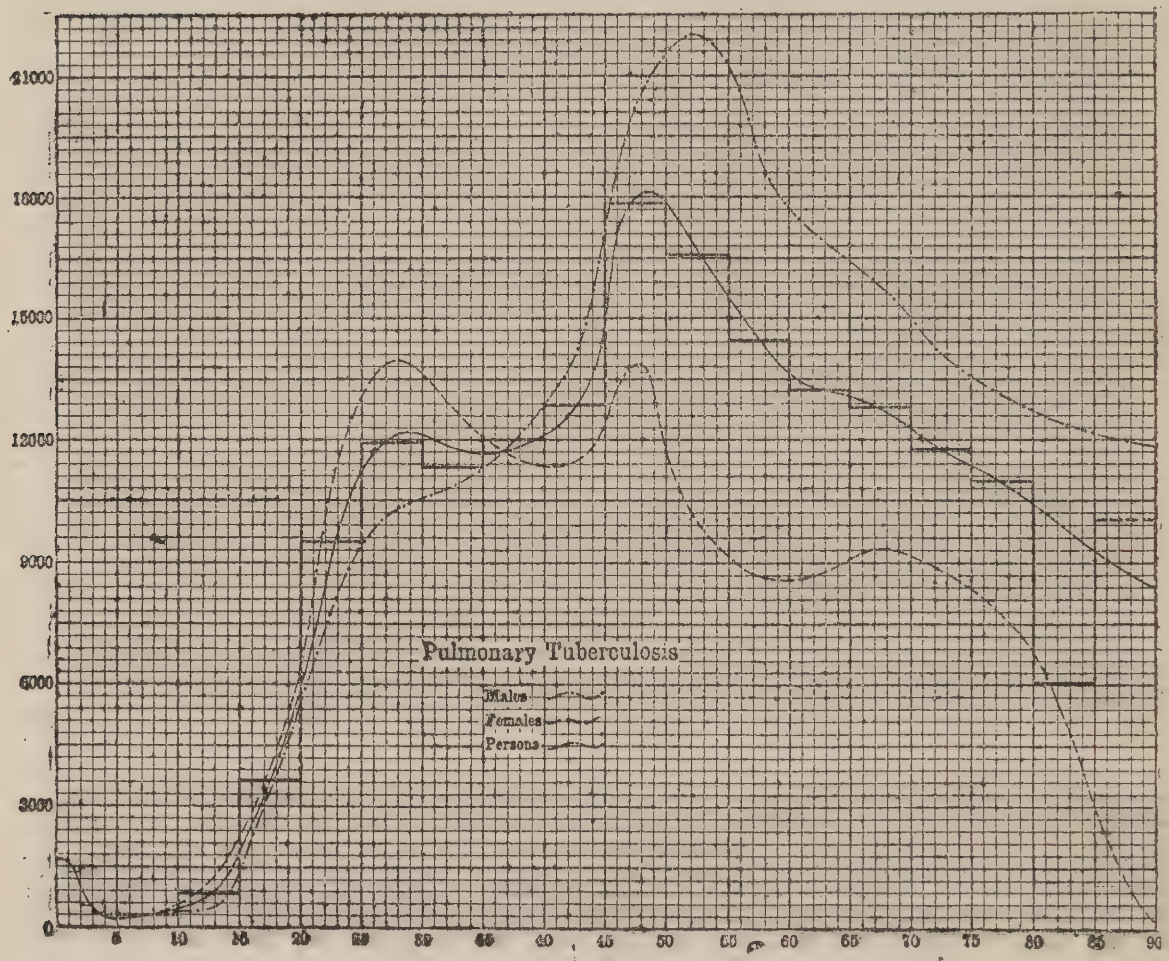
* The actual data give a dimorphic curve with maxima at 37 and 47 years of age.

The maximum frequency of death from cancer is for Males at about age 69 years, for Females at about age 67 years, and for Persons at about age 67 years. The frequency of death from cancer at different ages is shown on Graph No. 3, curves F.

COMMONWEALTH OF AUSTRALIA.—DEATH-RATES OF PERSONS OF DIFFERENT AGES, PULMONARY TUBERCULOSIS, 1907 TO 1910.

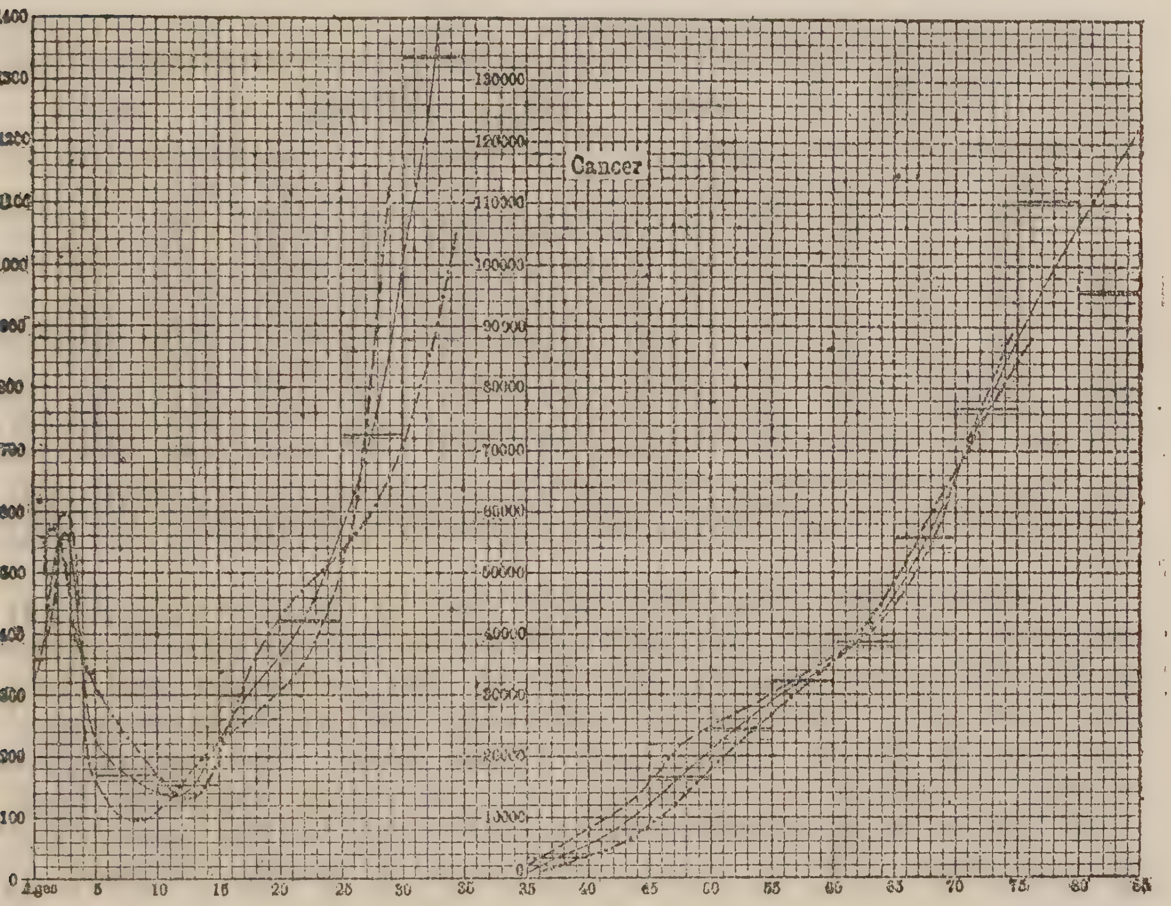
Graph No. 4.



Explanation of Graphs.—The curves represent the number of deaths per 10,000,000 of population at each age, the continuous line representing the number of deaths per 10,000,000 of total population, the broken line representing the number of female deaths per 10,000,000 of female population, and the dot and dash line representing the number of male deaths per 10,000,000 of male population. The base of each small square represents one year of age, and the vertical height a death-rate of 300 per 10,000,000. The figures on the left-hand margin represent the death rate per 10,000,000, those on the lower margin represent years of age.

COMMONWEALTH OF AUSTRALIA.—DEATH-RATES OF PERSONS OF DIFFERENT AGES, CANCER, 1907 TO 1910.

Graph No. 5.



Explanation of Graphs.—The broken line with dots — - — - — - — - — denotes the results for males; the plain broken line — — — — — denotes those for females, and the continuous line those for persons (both sexes combined).

The base of each small square represents one year of age, while the height represents a death-rate of 20 per 1,000,000 of population in the curve on the left of the diagram, and a death-rate of 2,000 per 1,000,000 of population in the curves on the right. The marginal figures on the left of the respective curves denote rates of mortality per 1,000,000 of population at the respective ages which are indicated in the lower margin of the diagram.

8. Death-rates from Pulmonary Tuberculosis and Cancer in each Age-Group.

The frequency of death for age-groups, expressed as the ratio to the total population, is not exactly comparable between one country and another, or the same country at different epochs, inasmuch as the distribution of population according to sex and age is by no means necessarily identical in the two cases. For this reason a further table is given showing the three death-rates in each age-group, viz., for males, for females, and for persons. This gives the ratio of the persons dying to the number living of the corresponding sex, in each age-group. From these results can be deduced the curve of death-rates according to age, the effect of the accidental element—the "constitution according to sex and age"—being eliminated. The following table furnishes the death-rates as defined :—

FREQUENCY of Deaths in age-groups from Pulmonary Tuberculosis and from Cancer per 1,000,000 (of corresponding sex and age-group) per annum, Australia, 1907–10.

Age-group.	Pulmonary Tuberculosis.			Cancer.			Age-group.	Pulmonary Tuberculosis.			Cancer.		
	M.	F.	P.	M.	F.	P.		M.	F.	P.	M.	F.	P.
0	165	171	168	38	34	36	35–39	1,216	1,184	1,202	205	498	334
1	166	107	137	57	43	50	40–44	1,398	1,141	1,288	561	1,127	803
2	67	38	53	46	59	53	45–49	2,089	1,366	1,784	1,314	2,187	1,683
3	36	32	34	41	37	39	50–54	2,160	1,006	1,651	2,272	2,684	2,452
4	10	37	23	31	37	34	55–59	1,906	869	1,443	3,140	3,315	3,218
5–9	30	34	32	24	10	17	60–64	1,681	870	1,322	3,789	3,974	3,871
10–14	53	117	84	14	17	15	65–69	1,619	921	1,313	5,763	5,339	5,577
15–19	336	392	364	34	26	30	70–74	1,409	852	1,179	7,600	7,860	7,687
20–24	776	1,124	950	48	37	42	75–79	1,315	809	1,097	10,889	11,259	11,049
25–29	1,028	1,367	1,193	60	86	73	80–84	696	489	603	9,929	9,118	9,563
30–34	1,067	1,200	1,129	88	194	134	85–	1,812	111	1,004	14,292	12,460	13,421

Note.—The death-rate in age-groups for "persons" as between two countries is only comparable when the group-masculinity is identical—that is, when in each age the relative number of males to females is the same.

It is obvious from the above table that, in the case of deaths from pulmonary tuberculosis, the curve showing the rate for males is, throughout its whole range, sensibly different to that for females, and the diagram shows this. The minimum rate, calculated for the same age-group, is at the age 3½ for females, and 4⅔ for males. The maximum rate similarly calculated is at about 52 or 53 for males, and 27 for females. In the case of cancer the difference is less striking, but the curve of death-rate calculated for the same age-groups, is totally different. The minimum death-rate for cancer for the same age-groups for males is at about 12 or 13 years of age, and for females at 7 or 8 years of age. The rate then probably continually increases. For "persons" the tuberculosis minimum rate, calculated for the same age-groups, is at about 4½ years, and the maximum at about 50, while in the case of "persons" dying of cancer, the minimum is at about 10.

The tuberculosis results given in the preceding table are shown in Graph No. 4, and the cancer results in Graph No. 5.

9. Conclusions.

The figures discussed indicate, as already stated, a clearly defined decrease in the rate of mortality from pulmonary tuberculosis, and a corresponding increase in that arising from cancer. To what extent either of these phenomena is due to improvement of diagnosis I have not attempted here to discuss, but, as far as my investigations have gone, I have been able to satisfy myself that the respective trends are real, and not merely apparent. It has I know been urged that the recorded decrease in pulmonary tuberculosis is due in part to the exclusion in recent years of tubercular diseases of non-respiratory organs, owing to more complete diagnosis and certification, and, on the other hand, that the recorded increase in cancer has been caused in part by the improvements in diagnosis in recent years, which have led to cases being classed as cancer which were formerly attributed to other causes. Apart from any other consideration, however, the remarkable regularity in the respective decreases and increases during the past thirty years suggests (or one might almost say, proves) that the main cause of variation is much more deeply seated, and that we have in operation in Australia a genuine decrease in the annual toll levied by the "White Plague" and an equally genuine increase in the ravages of cancer. In the former there is much cause for congratulation and much encouragement to press forward the work of prevention; in the latter there is grave cause for alarm. True, as indicated by the foregoing figures, the age incidence of the two diseases is entirely different, and whilst pulmonary tuberculosis claims its victims mainly from those in youth and in the prime of life, cancer is mainly a disease of advancing or advanced age. Economically, therefore, the substitution of cancer for pulmonary tuberculosis as a cause of death may be a gain; but why there should be any such substitution, or why suitable remedial measures for checking the increase of cancer should not be as effective as those which have produced such good results in the case of pulmonary tuberculosis, is a matter for the physician rather than for the statistician.

The following conclusions may be indicated :—

(i) Although deaths from tuberculosis and from cancer are quite dissimilar, in respect of the age at which death is most frequent, and in other respects, and appear also to be wholly unrelated, nevertheless deaths from both diseases combined remain nearly constant both in Australia as in England and Wales.

(ii) The rate of deaths of males, though considerably higher than that for females, is diminishing more rapidly in the case of tuberculosis; the rates are nearly equal for cancer, and are increasing practically at the same but a smaller rate than in the case of tuberculosis. Thus for both diseases taken together there is a greater frequency in deaths, but also a more rapid improvement, in the case of males.

(iii) There is a fairly well defined fluctuation of frequency of death in the several months of the year for each disease.

(iv) The frequency according to age exhibits the following features, viz. :—

Pulmonary Tuberculosis.—Increasing values after 5 years of age to a maximum, which, for "persons," is probably about age 27 years, and a decline thereafter. The maximum for males is probably about age 42 years, and for females probably about age 26½ years.

Cancer.—Similar variation, with a maximum for "persons," however, at about age 67 years; for males at about age 69 years, and for females at about age 67 years.

(v) The death-rate for age-groups exhibits the following characteristics, viz :—

Pulmonary Tuberculosis.—Increase after 5 years of age to a maximum for persons at age 47, and decline thereafter.

Cancer.—Low rates at early ages, succeeded by continuously increasing rates as age advances.

THE IMPROVEMENT IN INFANTILE MORTALITY: ITS ANNUAL FLUCTUATIONS AND FREQUENCY ACCORDING TO AGE IN AUSTRALIA.

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SYNOPSIS.

1. Introductory.
2. Secular fluctuation of infantile mortality.
3. Significance of the secular changes in infantile mortality.
4. Measurement of the improvement in infantile mortality.
5. Magnitude of deviation from mean conditions.
6. Masculinity of infantile mortality.
7. Frequency of infantile mortality according to age for the first month of life.
8. Average duration of infant life.
9. Diminution of infantile mortality in successive months.
10. Conclusions.

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1. Introductory.

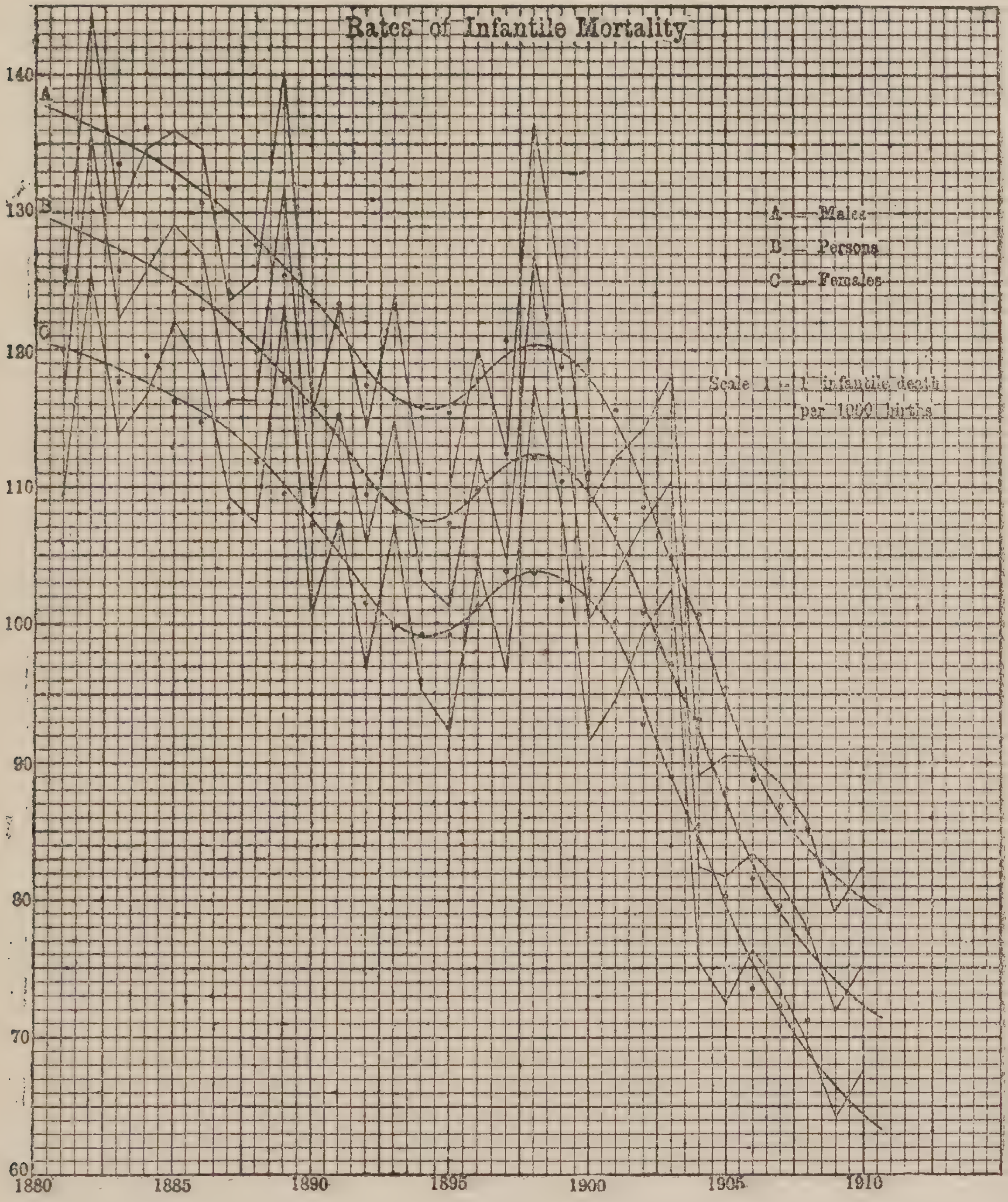
Comment has been made from time to time on the rapid diminution of the birth-rate in Australia. In this connection it is well to remember, however, that what is important in a community is not so much the *actual* as the *effective* birth-rate. This last is to be based, not merely on the number born, but on those who survive for any particular period chosen for the purposes of comparison. In order, therefore, to ascertain whether birth-rate is satisfactory, one must also take account of the whole course at least of infantile mortality. This is but part of a larger question, viz., the significance for life aggregates of any variations in the frequency of death at each age, and the economic and social meaning of such variations. This article deals with the secular trend of the rate of infantile mortality; its annual fluctuations; the frequency of deaths of infants according to age, and other matters necessary to the determination of those.

2. Secular Fluctuation of Infantile Mortality.

In the following table of the rate of infantile mortality, the rate is the ratio of the number of deaths of children of under 1 year of age to the number of births in the *equivalent* year, multiplied by 10,000. It thus purports to give the number of deaths in the year per 10,000 infants born. In regard to what is called the *equivalent year*, it is shown hereinafter that all children who die in the first year of their existence live on the average for 99·3 days, this result being based on an investigation for the years 1909 and 1910. The registration of children, based upon an investigation for the first quarter of the present year, shows that, on the average, registration is 38·2 days later than birth. The difference, therefore, is 61·1 days, or say 2 months, and the equivalent year, therefore, in which the births occur, and to which the infantile mortality must be referred for the determination of its rate, is therefore approximately 60 days, or say two months earlier than the calendar year in which the infantile deaths actually occur, the latter being of course registered immediately. Since registration of births (and also births themselves) are fairly uniform throughout the year, it will be sufficiently accurate to add one-sixth of the births of the calendar year preceding any year in question, and subtract one-sixth of the births of the year itself to obtain the number of births in the equivalent year. Then the infantile deaths,

COMMONWEALTH OF AUSTRALIA.—RATES OF INFANTILE MORTALITY, 1881 TO 1910.

Graph No. 6.



Explanation of Graphs.—The base of each small square represents an interval of half a year, while the vertical height represents an increment in infantile death rate of one per 1,000 births, an infantile death rate of 60 per 1,000 births being taken as base line for the diagram. The figures on the left-hand margin of the diagram represent rates of infantile mortality per 1,000 births, while those on the lower margin denote calendar years. The black dots represent a yearly succession of quinquennial means, from which the general trend of the rate of infantile mortality has been ascertained; the upper curve (A) showing the trend for males, the lower (C) for females, and the middle curve (B) for persons (both sexes combined).

constituting the numerator in the ratio, are those occurring in the calendar year itself for which the rate is formed. That this process is abundantly accurate is obvious from the fact that the actual birth-registration for the months of November and December are about 97 per cent. of the number which would be given if these months gave the average for the year. The rates, found as explained, for the period 1881 to 1910 are as follows :—

INFANTILE Mortality Rate in Australia, 1881 to 1910.

Year.	Mortality per 10,000 births.			Masculinity of infantile mortality.		General trend in rates of infantile mortality.		
	Males.	Females.	Persons.	Observed.	Computed.	Males.	Females.	Persons.
(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
1881	1,232	1,095	1,165	1,127	1,136	1,372	1,203	1,293
1882	1,446	1,265	1,357	1,143	1,136	1,363	1,195	1,284
1883	1,302	1,138	1,222	1,144	1,137	1,353	1,186	1,274
1884	1,348	1,168	1,260	1,154	1,137	1,342	1,176	1,263
1885	1,360	1,221	1,292	1,114	1,138	1,330	1,166	1,251
1886	1,348	1,189	1,271	1,134	1,139	1,316	1,155	1,238
1887	1,235	1,091	1,164	1,132	1,140	1,300	1,140	1,222
1888	1,251	1,072	1,164	1,167	1,142	1,281	1,122	1,203
1889	1,400	1,234	1,319	1,134	1,143	1,260	1,101	1,182
1890	1,152	1,009	1,082	1,142	1,145	1,237	1,078	1,159
1891	1,232	1,074	1,155	1,147	1,147	1,212	1,053	1,135
1892	1,142	970	1,058	1,177	1,149	1,188	1,024	1,108
1893	1,240	1,072	1,149	1,157	1,151	1,167	998	1,086
1894	1,107	952	1,031	1,163	1,154	1,158	993	1,076
1895	1,099	921	1,012	1,193	1,156	1,161	997	1,079
1896	1,202	1,045	1,126	1,150	1,159	1,177	1,012	1,096
1897	1,126	967	1,048	1,164	1,162	1,196	1,031	1,115
1898	1,364	1,175	1,272	1,161	1,165	1,204	1,038	1,125
1899	1,246	1,085	1,167	1,148	1,168	1,198	1,034	1,117
1900	1,086	915	1,002	1,187	1,172	1,181	1,019	1,097
1901	1,122	947	1,037	1,184	1,176	1,145	993	1,062
1902	1,142	997	1,071	1,145	1,180	1,103	946	1,019
1903	1,183	1,025	1,105	1,154	1,184	1,053	892	970
1904	891	756	825	1,179	1,188	1,000	843	921
1905	908	725	819	1,253	1,192	947	796	873
1906	906	764	836	1,186	1,197	900	753	827
1907	886	737	814	1,203	1,202	863	719	792
1908	857	698	780	1,227	1,207	838	691	765
1909	790	642	718	1,230	1,212	818	665	741
1910	820	677	751	1,211	1,217	802	646	723

The data in the above table are represented on Graph No. 6, curves A, B, and C, see zig-zag lines and the smoothed curves.

The results in Columns (2), (3), and (4) of the above table cannot be represented by any simple formula, and point to the fact that while there is a general trend in the direction of a lowering of infantile mortality, it is by no means continuous. This fact we proceed to consider.

3. Significance of the Secular changes in Infantile Mortality.

In the above table, columns (2), (3), and (4) give the deduced mortality rates per 10,000 births for males, females, and persons respectively. It is hardly possible to grasp the full significance of these from a perusal of the table itself; a diagram has therefore been prepared showing the actually observed rates, and by straight lines joining these points, the fluctuations of the actual rates may readily be followed. The striking feature of these variations in the infantile mortality rates is that the larger fluctuations are sensibly the same for the two sexes; but minor fluctuations show differences. This is to be expected, because the larger fluctuations are doubtless the expression, in infantile mortality, of specially adverse or of specially favourable climatological conditions. These remarkable correspondences and minor diversities of the fluctuations, as between the two sexes, are readily seen by comparing the upper zig-zag line for male rates of infantile mortality with the lower for female rates. The middle zig-zag line, which is nearly, though not exactly, a mean between the two referred to, represents the rate for persons, *i.e.*, for males and females combined.

To analyse such results as are indicated in the diagram, we observe at once that individual years are affected by special characteristics. For example, years in which there was great and continuous heat, with perhaps marked humidity, would be years characterised by a high rate of infantile mortality, because the conditions for serious gastro-intestinal disturbance would be present, and this contributes largely to the result. Similarly, any years which are characterised by either specially untoward or specially favourable conditions of any kind whatever must deviate in one or the other direction from what may be called the *general trend* of the facts. It is obvious that the deviations referred to tend to hide the general trend; for example, an exceptionally bad year would quite mask the effect of general improvement in child-hygiene, more rational feeding, and better medical treatment, though the latter must undoubtedly have reduced the number of deaths from what they would have been but for these favourable factors.

In order to obtain the general trend of any facts, it is necessary to combine a sufficient number of results to balance the deviations in either direction. I have, therefore, by successive quinquennial groupings, shifting the mean year only one year at a time, endeavoured to deduce the general trend, and by this means to discover the steadily progressive improvement in the rate of infantile mortality, which is so remarkable a feature of Australian Vital Statistics.

The points obtained by such quinquennial grouping, shown by dots on the diagram, give nearly a regular curve, because what may be called the accidental deviations of individual years on either side tend to balance each other, and, as a matter of fact, practically do so. That this is to be expected can, *a priori*, be seen by an inspection of the fluctuations. A smooth curve was then drawn through these quinquennial mean places, and this may be taken to represent the general trend, free from all annual accidental variations, in fact from all variations which do not persist over a number of years.

In this way the curves have been drawn for the rates for males, females, and persons, and any general deductions may more readily be made from these than from the original data. The numerical values corresponding to these curves are given in columns (7), (8), and (9) of the preceding table. (See the smoothed curves A, B and C on Graph No. 6.)

4. Measurement of the Improvement in Infantile Mortality.

A numerical estimate of the improvement in infantile mortality obviously should be referred not to abnormal but to normal or average conditions. Hence it is preferable to deduce it from values of the general trend of the infantile mortality rather than from the results for particular years, deviating more or less strikingly from the general trend. The curve representing this shows an improvement from 1881 to 1894, a retrograde development from 1894 to 1898, and then a more decided improvement, continuing up to the present time. This is also evident from an inspection of the figures in column (9) of the preceding table.

To give numerical expression to this we may arbitrarily take the rate for 1881 as the basis of comparison, and indicate for any subsequent year the improvement by showing its rate as a percentage on that for 1881. In this way we obtain :—

Improvement in Australian Infantile Mortality in 30 years.

Year.	Males.	Females.	Persons.
1881	100	100	100
1888	93·4	93·3	93·0
1894	84·4	82·5	83·2
1898	87·8	86·3	87·0
1901	83·5	82·5	82·1
1905	69·0	66·2	67·5
1910	58·5	53·7	55·9

The improvement indicated in this table is very striking, viz., a reduction of male infantile deaths as in 1881 to only 58·5 per cent., and female infantile deaths to 53·7 per cent. Thus we see, moreover, that it is somewhat more difficult to secure the same degree of reduction of mortality in males; and this is evident for each year for which results are given in this last table. This fact is also disclosed in the figures showing the progression of the masculinity of infantile mortality (see section 6 hereinafter), and gives numerically nearly the same result.

5. Magnitude of Deviation from Mean Conditions.

It will be sufficient here to discuss the deviation from mean conditions for persons, leaving the more thorough analysis to a later date when the necessary meteorological data are available to be taken into account. The general trend curve can be assumed to represent mean conditions.

Deviations in Individual Years from Mean Conditions.

CASES of Infantile Mortality in 10,000 Births.

1881	−128	1886	+ 33	1891	+20	1896	+ 30	1901	− 25	1906	+ 9
1882	+ 73	1887	− 58	1892	−50	1897	− 67	1902	+ 52	1907	+22
1883	− 52	1888	− 39	1893	+63	1898	+147	1903	+135	1908	+15
1884	− 3	1889	+137	1894	−45	1899	+ 50	1904	− 96	1909	−23
1885	+ 41	1890	− 77	1895	−67	1900	− 95	1905	− 54	1910	+28

Deviations = observed rate minus general trend rate: Column (4) − column (9) in first table.

The average deviation is 57·8 in an average of roughly 1,100 deaths or 10,000 births, and the extreme variations are only -128 to $+147$. That is to say, what we have called the general trend differs from the observed results on the average about $\pm 5\frac{1}{4}$ deaths per 100, and never differs more than $\pm 13\frac{1}{2}$ per 100. We must, therefore, regard the increase of infantile mortality from 1894 to 1898, as something more than an accidental variation.

6. Masculinity of Infantile Mortality.

In column (5) of the table in section 2 is shown the ratio of infant males per 10,000 male births dying within 1 year, to infant females per 10,000 female births dying within 1 year, that is to say, when the number of births of each sex are equal. The ratio is shown by the number of males per 1,000 females and reveals a curious progression. This ratio may be called the masculinity of infantile mortality. If we form its quinquennial averages, we get for the six quinquennia the following figures, viz. :—

1,136, 1,142, 1,167, 1,162, 1,183, 1,211,

which is a sufficiently clear indication of a steady progression. By appropriate methods of solution, I find that the masculinity of infantile mortality (denoted in the formula hereunder by D) can be expressed with quite remarkable precision as a function of the data. The formula I have deduced is—

$$D = 1{,}136 + 0{\cdot}09\,(T - 1880)^2;$$

in which T is the year for which the masculinity is desired. The ratios computed by this formula are shown in column (6) for comparison with the actually observed ratios, and the coincidence is certainly remarkably close. The assumption of a linear change, however (an equal change year by year), instead of one progressing as the square of the time, will not fit the data.

It is not a little remarkable that, with an improvement in infantile mortality, both sexes are not equally affected, that is to say, although the mortality was relatively less with female than with male infants in 1881, with the lapse of time there has been a more marked improvement in female infantile, than in male infantile, mortality. In other words, better hygiene, feeding, and medical care have not been so successful with male as with female infants, as was indicated previously in section 4.

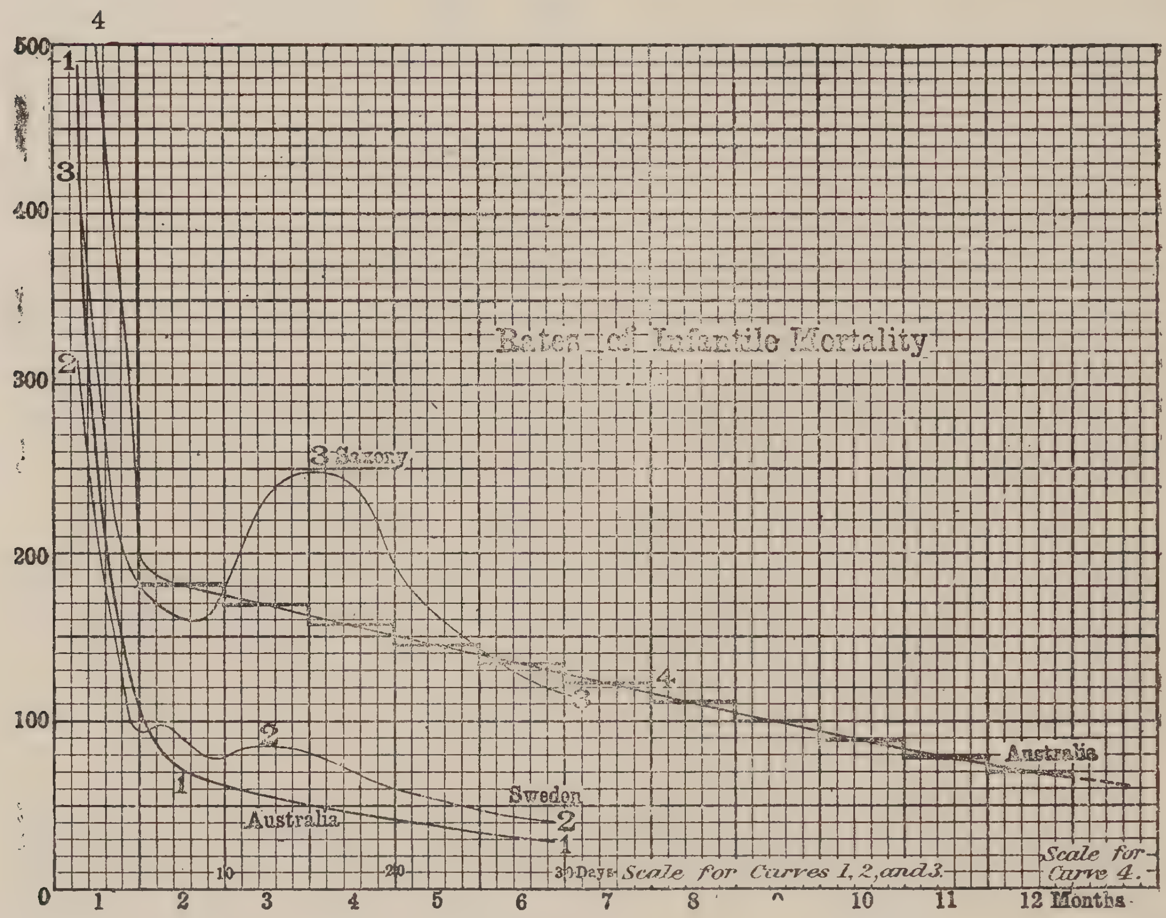
The actual number of male lives saved is, however, somewhat greater that the actual number of female lives, being 570 per 10,000 births in the case of males, and 557 per 10,000 births in the case of females when the experience of 1910 is compared with that of 1881.

7. Frequency of Infantile Mortality according to age for the First Month of Life.

Out of a given number born, the number who survive to each successive day up to the thirtieth day, reveals the characteristic features of infantile mortality, viz., the way in which death reduces the number at each age, or day by day. This is obvious, since the diminishing remainders formed by subtracting the deaths each day, give the number of survivors from which the quota of decedents on the next day is drawn.

RATES OF INFANTILE MORTALITY.—AUSTRALIA, SWEDEN, AND SAXONY.

Graph No. 7.



Explanation of Graphs.—For curves 1, 2, and 3, the base of each small square represents one day, while the height represents ten infantile deaths per day from 100,000 births. For these curves the left-hand marginal marking represents the number of infantile deaths in successive days from 100,000 births.

For curve 4, the base of each large square (comprising twenty-five small squares) represents one month, while the height of each of the component small squares represents ten infantile deaths per day from 1,000,000 births. For this curve the left-hand marginal marking represents number of infantile deaths per day from 1,000,000 births.

It has been possible to carry out an investigation of this kind for Australia only for the years 1909 and 1910, and the results, owing to loose statements about the duration of life, are not quite satisfactory. In the original figures the somewhat large number for deaths at 7 days, and the abnormally large numbers for deaths at 14 and 21 days, are due to the fact that the age at death is loosely stated to be 1, 2, or 3 weeks, instead of the exact number of days. Hence many of these must be assigned to the days previous and subsequent to the exact multiple of 7 days. It may be added, further, that from a comparison of the frequency of death between 1 and 2, 2 and 3 months, &c., it is also evident that the number of deaths is too few for the closing days of the month.

In order to get some idea of the probable curve, the excess over the mean of the preceding and following days (6th and 8th, 13th and 15th, &c.) has been assumed (somewhat arbitrarily it must be admitted) to be in the ratio of the numbers 1, 3, 5, 7, 9, 7, 5, 3, 1, the proportion 9/41 being added to the day itself, 7/41 to each of the two contiguous days, 5/41 to each of the next days but one, and so on.

The numbers of deaths stated for the first and second days are undoubtedly incorrect, and to obtain a probable result, the figures for Victoria, South Australia, and Western Australia, in which the numbers of deaths under twenty-four hours are greater than from twenty-four to forty-eight hours, were taken as correct. In New South Wales, Queensland, and Tasmania, deaths occurring during the first twenty-four hours are evidently often registered as those of persons one day old. The ratio of deaths occurring during the first twenty-four hours to those occurring during the second twenty-four hours was found to be as 16 : 7 in the first-mentioned States. Final figures were obtained by halving the number of deaths at one day, and adding $\frac{1}{2} + \frac{1}{60}$ of this number, to the number of deaths of children under one day, that correction being roughly indicated as necessary on account of the diminution of frequency with the increased duration of life.

Further, it was necessary, in order to obtain the normal frequency toward the end of the month, to use the evidence derivable from the group results for the successive months, according to a suitable scheme.

The curve was then smoothed, giving the numbers in the final column. This distribution, through somewhat arbitrary, has nevertheless a high degree of probability, and the smoothed curve gives some idea of the probable infantile mortality day by day.

An allowance for variation in the number of births for the two years was found to be unnecessary. The ratio of deaths at each specified age to the total births has therefore been taken as the frequency.*

The final figures give the probable daily mortality per million births.

For comparisons of frequency, results are available for Sweden for 1901–5, and for Saxony for 1891–1905. (See article by Dr. Med. E. Roesle, "Die Sterblichkeit im ersten Lebensmonat; Zeit. für Soz. Mediz., Leipzig." Bd. V, p. 175, 15 April, 1910.)

Number of Deaths per diem of 1,000,000 *children born alive in Australia, Sweden, and Saxony.*

No. of day.	Australia (smoothed). 1909–1910.	Sweden. 1901–1905.	Saxony. 1891–1905.	No. of day.	Australia (smoothed). 1909–1910.	Sweden. 1901–1905.	Saxony. 1891–1905.	No. of day.	Australia (smoothed). 1909–1910.	Sweden. 1901–1905.	Saxony. 1891–1905.
1	8,403	6,320	9,830	11	599	860	1,860	21	410	560	1,900
2	4,886	3,130	4,260	12	577	830	2,140	22	394	580	1,710
3	2,581	2,160	3,240	13	556	860	2,360	23	379	550	1,620
4	1,733	1,530	2,340	14	535	830	2,420	24	364	530	1,560
5	1,234	960	1,890	15	515	840	2,480	25	349	510	1,460
6	975	960	1,750	16	496	770	2,450	26	335	470	1,400
7	794	1,000	1,700	17	478	730	2,490	27	321	430	1,340
8	717	920	1,630	18	460	730	2,420	28	308	440	1,250
9	677	830	1,580	19	443	660	2,290	29	295	410	1,210
10	647	730	1,710	20	426	630	2,040	30	282	410	1,340
Totals 0–10	22,647	18,540	29,930	Totals 11–20	5,085	7,740	22,95	Totals 21–30	3,437	4,890	14,790

Grand Totals—Australia, 31,169; Sweden, 31,170; Saxony, 67,670.

The results in the above table are shown on Graph No. 7, see curves 1, 2, and 3.

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* NOTE.—This corresponds to the d_x column in actuarial tables.

It will be seen from the above that after the sixth day the conditions in Australia are very much better than in either Sweden or Saxony. In both the latter countries the numbers of deaths show a recrudescence of the mortality rate. In Sweden the fifth and sixth days are a first minimum, the seventh day is higher; there is a second minimum at ten days, a second maximum about the thirteenth day. In Saxony, for which the curve is far more regular, the first minimum is the ninth day, and the maximum following it the seventeenth day. There is no indication of a similar feature in Australia, that is to say, the probability of death falls for each successive day. At no part of the thirty days is there any sign of increasing liability to death.

A comparison of the grand totals shows that at the end of thirty days, the relative aggregate loss of infant life is identical for Sweden and Australia, and both are less than one-half of the relative loss for Saxony.

In Graph No. 7 the curves 1, 2, and 3 show the change in the number dying daily out of 100,000 births (not 1,000,000 as in the table) for Australia, Sweden, and Saxony, respectively, for the years mentioned in the table. The curves have been "smoothed" to show the probable trend with large numbers, but nevertheless still represent very exactly the tabular figures. It is at once evident from the diagram that Australian infantile mortality exhibits no sign of the recrudescence of the rate as is shown so markedly in Saxony, and less strikingly in Sweden. Possibly greater care by Registrars of Deaths as to the exact length of the intervals between birth and death would give some indication of a similar fluctuation in the death-rate: it has already been pointed out that the term "week" is used loosely in Australian registration, and owing to this the tabulation is to some extent vitiated.

A marked improvement in this respect would be effected if the registers showed "*date of birth*" instead of "*age*."

8. Average Duration of Infant Life.

The significance of the rapidity with which children die during the first month of existence, and also during successive months up to twelve, is shown by indicating the average duration of infant life for all those who die during the period terminating at the end of the first, the second, and so on to the end of the twelfth month.

The results are as follows :—

Average Duration of Days of Infant Life, Australia, 1909–10.

CHILDREN dying up to end of month 1 to 12 after birth :—

Month	1	2	3	4	5	6	7	8	9	10	11	12
Average duration (days) ...	7·3	13·1	20·7	29·7	38·5	47·9	57·3	66·4	75·4	83·8	91·7	99·3

Incidentally it may be remarked, that we see from this that to properly determine the rate of infantile mortality in Australia, the yearly aggregate of infantile deaths should be referred not to the births in the same year, but to those of a year ending 99·3 days earlier.

9. Diminution of Infantile Mortality in successive months.

In all countries the very high rate of infantile mortality lasts only for the first month, and then greatly diminishes. This is seen in the following figures for Australia for equalised months of 30·42 days each :—

Average Rate of Death (per diem) per 1,000,000 *Births in Australia, in successive months from Birth,* 1909–10.

Month	1	2	3	4	5	6	7	8	9	10	11	12
Deaths per diem (smoothed curve)	1,047	181	169	157	145	133	122	111	100	89	79	70

The data in this table are represented on Graph No. 7, curve 4.

To compare these rates of infantile mortality with those of the United Kingdom, New Zealand, &c., we form the groups 1 to 3 months, 4 to 6 months, and 7 to 12 months., as in the following table :—

Mean Rate of Infantile Mortality in successive periods from Date of Birth, per 10,000 *Births.*

Country.	Period.	1st to 3rd month.			4th to 6th month.			7th to 12th month.			1st to 12th month.		
		M.	F.	P.	M.	F.	P.	M.	F.	P.	M.	F.	P.
Australia ...	1903–1910	513	402	459	172	154	164	213	190	201	898	746	824
New Zealand ...	1901–1909	486	379	434	154	126	140	159	140	150	799	645	724
England and Wales ...	1901–1909	756	583	672	276	235	255	395	347	372	1,427	1,165	1,299
Scotland ...	1901–1908	695	543	620	237	195	217	365	324	345	1,297	1,062	1,182
Ireland ...	1901–1908	616	493	556	182	159	171	255	224	240	1,053	876	967

In the above table the rates given are the means of the annual rates for the periods indicated. It will be noticed that New Zealand is unequivocally in the most favourable position; with Australia next; then Ireland, Scotland, and England and Wales.

10. Conclusions.

The general result shows the following, viz. :—

(i) That Australia occupies a very favourable position as regards infantile mortality as compared with the United Kingdom. That New Zealand results are distinctly better is probably due to the fact that her aggregations of population are smaller.

(ii) The general trend of infantile mortality does not follow any simple law, and improvement, while general throughout, was not continuous from 1881 to the present time.

(iii) Individual years differ in a very marked manner from the general trend, and disclose the fact that when the differences are large, they are exhibited by each sex, and hence show that particular years are distinctly favourable or unfavourable to infant life.

(iv) It would appear also that adverse, and probably also beneficial conditions may persist for several successive years.

(v) An investigation, taking the climatological factors into account, of the departure of individual years from the general trend would appear to be desirable.

This, however, has not yet been attempted.

THE SECULAR AND ANNUAL FLUCTUATIONS OF DEATHS FROM SEVERAL DISEASES IN AUSTRALIA.

SCARLET FEVER, MEASLES, WHOOPING-COUGH, DIPHTHERIA AND CROUP, TYPHOID, DIARRHŒA AND ENTERITIS AND DYSENTERY, AND THE FREQUENCY OF DEATH ACCORDING TO AGE OF THIS LAST.

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SYNOPSIS.

1. Introduction.
2. Secular fluctuation of death-rates for the several diseases; each sex.
3. Secular fluctuation of death-rates for both sexes together.
4. Masculinity of deaths by various diseases.
5. Frequency of deaths according to sex and age, diarrhœa and enteritis, and dysentery.
6. Death-rates from diarrhœa and enteritis, and dysentery according to age-groups.
7. Monthly variation in deaths from various causes.
8. Conclusions.

1. Introduction.

The annual frequency of death from the diseases mentioned in the title of this paper can be given for Australia for the thirty years between 1881 and 1910, deaths in Western Australia being included, however, only from the beginning of 1897, when they were first available in full detail. As the numbers for the State mentioned were very small, the result is not in any way prejudiced, and may be regarded as perfectly applicable to the whole of Australia from 1881. The fluctuations in the frequency from month to month have had perforce to be compiled from the records of 1907 to 1910 inclusive, since the necessary data were not earlier available in a complete form.

The frequency of death with age had also to be determined from the records of the same four years. The diseases included under the several headings are those which are embraced under the numbers indicated, according to the International Nosological Classification (1900), and are as follows:—

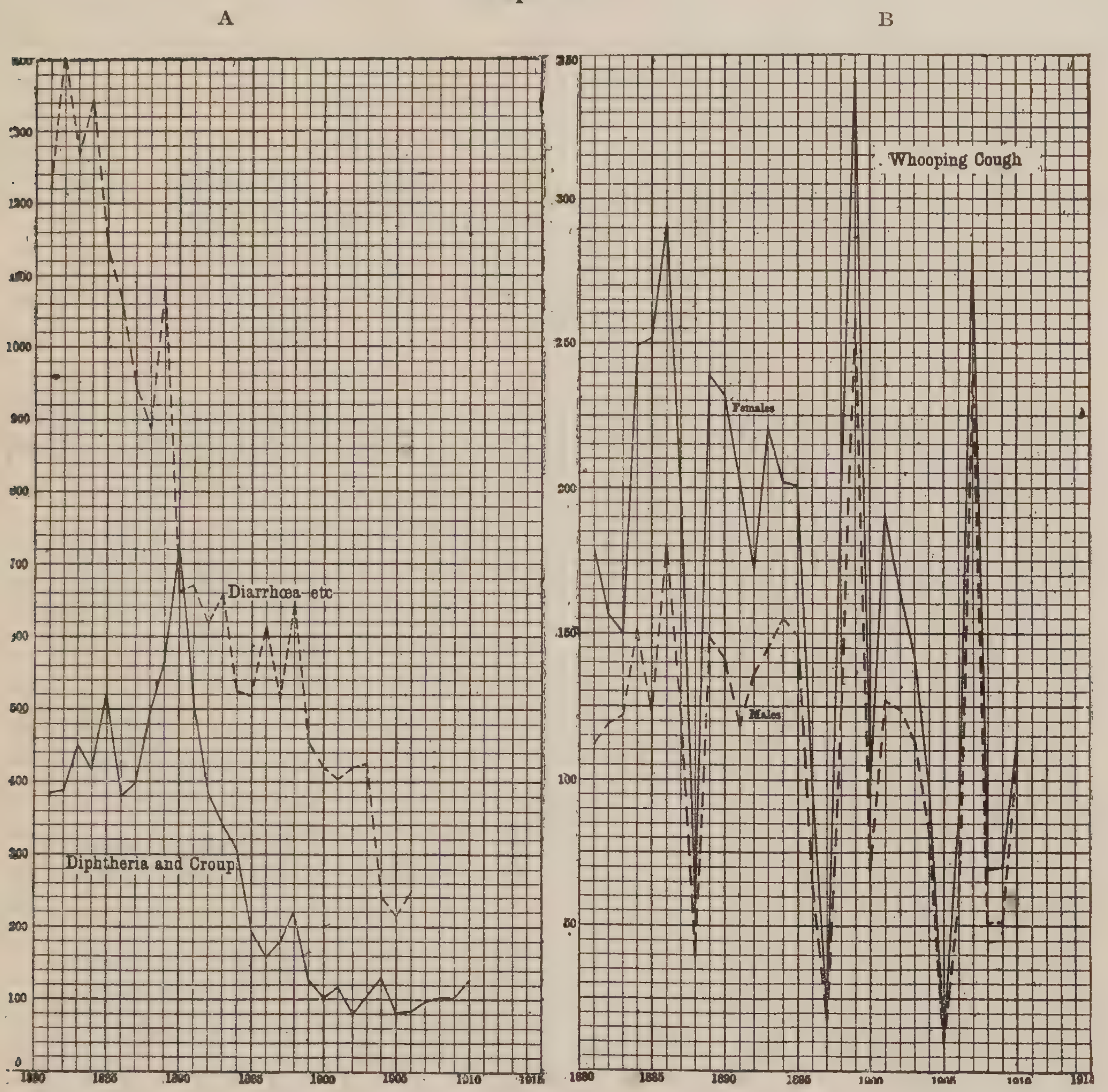
> *Scarlet Fever*, No. 7; *Measles*, No. 6; *Whooping Cough*, No. 8; *Diphtheria and Croup*, No. 9; *Typhoid*, No. 1; *Diarrhœa and Enteritis, and Dysentery*, Nos. 105, 105A, and 106, and No. 14.

2. Secular Fluctuation of Death-rates from the several Diseases, each sex.

The death-rates per annum per million persons of the corresponding sex are given for males and females. The results for the two sexes are shown side by side for convenience of comparison. The quinquennial averages given are simply the means of the rates for the quinquennia, and are not the weighted means; the two, however, are sensibly equal. The results for "persons" can be readily calculated by weighting according to the masculinity column, following the column of years.

COMMONWEALTH OF AUSTRALIA.—DEATH-RATES FOR DIARRHŒA, &c., FOR DIPHTHERIA AND CROUP, AND FOR WHOOPING COUGH, 1881 TO 1910.

Graph No. 8.



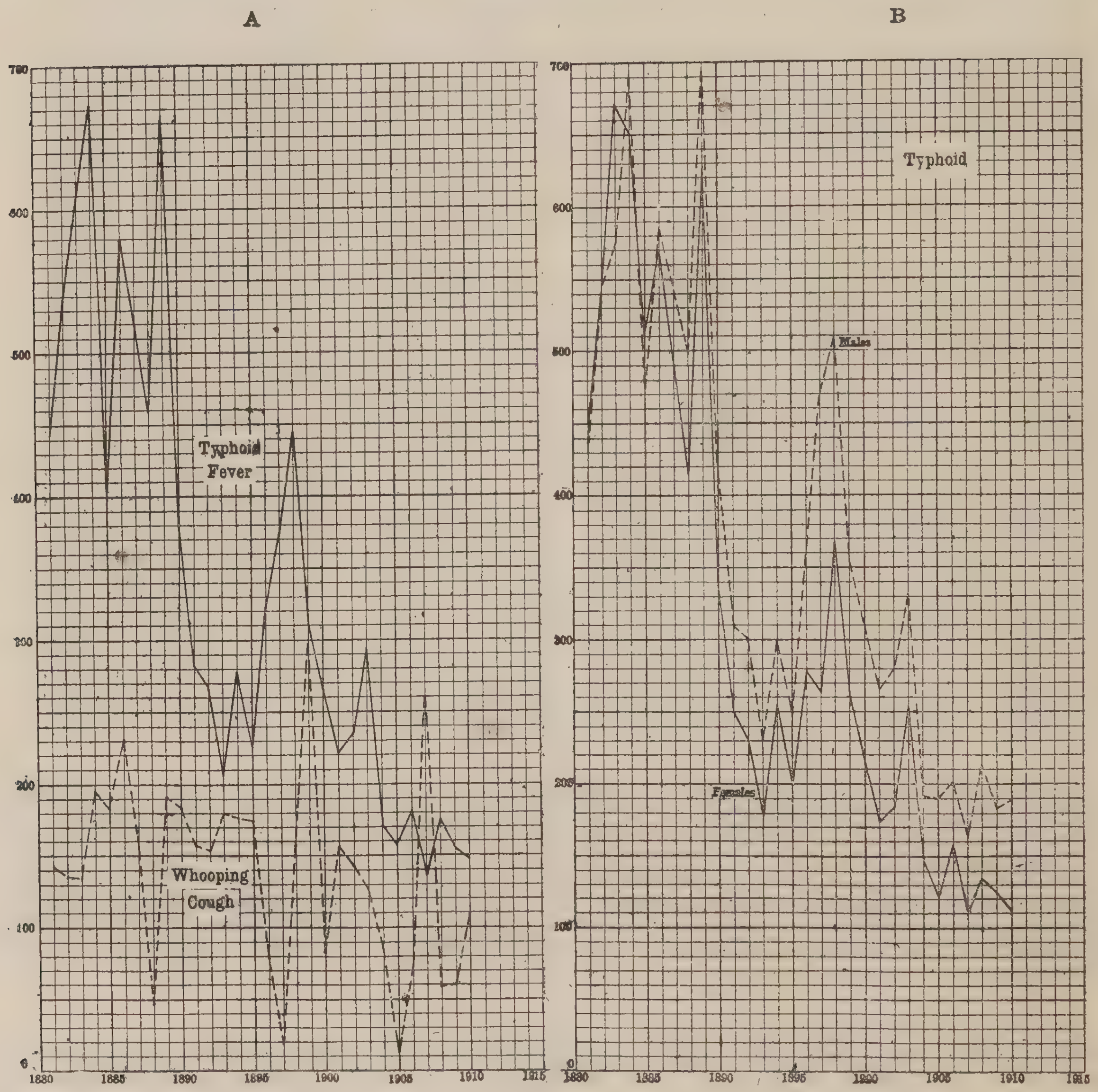
Explanation of Graphs.—The diagram on the left relates to death-rates of persons (males and females combined) from diarrhœa, &c., shown by a broken line, and from diphtheria and croup, shown by a continuous line; and that on the right relates to the male and female death rates (separately shown) from whooping-cough, the broken line denoting results for males and the continuous line those for females. In both cases the base of each small square represents one calendar year, while the vertical height in the diagram on the left represents a death-rate of 20 per 1,000,000 of population, and in that on the right a death-rate of 5 per 1,000,000 of male and female population, respectively. The figures on the left margin of each diagram are death-rates per 1,000,000 of population; those on the lower margins are calendar years.

This gives the number of males to 1,000 females in the general population. It has not been thought necessary to compute the results for each of the diseases, for the reasons that in the case of whooping-cough and typhoid the progression of the rates differs for each sex. With scarlet fever, measles, diphtheria and croup, and diarrhœa and enteritis, and dysentery, however, there is no special tendency dependent on sex, and consequently all differences from year to year may properly be regarded as quite accidental. Hence the annual rates for "persons" show the general trend better than the rates for either sex. The results for persons are consequently brought together in a second table.

For the same reason in the case of whooping-cough and typhoid, two graphs for each are furnished on the diagram, viz., one showing the results for males, the other for females; but in the case of the other diseases only one graph for each disease is given, viz., for "persons." For facility of reference, graphs for "persons" are also furnished in the case of typhoid and whooping-cough.

COMMONWEALTH OF AUSTRALIA.—DEATH-RATES FOR TYPHOID FEVER AND WHOOPING COUGH, 1881 TO 1910.

Graph No. 9.



Explanation of Graphs.—In the diagram to the left, both results are for persons (both sexes combined), the upper or firm line denoting the mortality for typhoid fever; the lower or broken line, the mortality or whooping-cough. In the diagram to the right, the results shown by broken lines indicate the mortality from typhoid for males; and the firm line shows the mortality from typhoid for females.

The base of each small square represents one calendar year, while the height represents a death-rate of 10 per 1,000,000 of mean population. The left-hand marginal marking represents death-rates per 1,000,000 of population in each case; while the figures on the lower margin are the calendar years at quinquennial intervals.

Rates of Death per annum per 1,000,000 *of the corresponding sex from various Diseases, Australia,* 1881–1910.

Year.	Masculinity.	Scarlet Fever.		Measles.		Whooping Cough.		Diphtheria and Croup.		Typhoid.		Diarrhœa and Enteritis, and Dysentery.	
		M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
1881	1,173	75	82	82	90	112	179	369	402	437	450	1,225	1,218
1882	1,174	71	68	22	38	119	156	352	428	545	537	1,472	1,335
1883	1,177	53	54	52	51	122	150	452	452	574	672	1,329	1,198
1884	1,182	92	143	148	192	151	249	395	440	692	650	1,576	1,067
1885	1,181	51	76	37	45	123	252	501	543	475	511	1,171	1,104
Mean (1–5)	1,177	68	85	68	83	125	201	414	453	544	564	1,355	1,184
1886	1,180	33	60	11	12	180	291	396	361	586	571	1,082	1,045
1887	1,177	32	31	48	46	123	196	368	435	542	492	981	983
1888	1,173	48	56	79	97	41	56	486	506	498	414	899	872
1889	1,166	34	38	9	14	149	239	537	594	697	628	1,114	1,045
1890	1,166	54	79	2	3	141	232	677	779	416	338	673	649
Mean (6–10)	1,172	40	53	30	34	127	203	492	535	548	489	950	919
1891	1,153	27	36	5	3	118	203	482	551	309	250	717	617
1892	1,145	29	35	1	1	136	172	369	403	301	230	645	591
1893	1,137	52	78	389	380	145	220	313	373	230	178	688	626
1894	1,127	56	74	93	106	155	202	276	342	299	255	556	487
1895	1,117	28	33	1	3	149	201	183	206	249	199	551	482
Mean (1–5)	1,136	36	51	98	99	140	200	325	375	278	222	631	561
1896	1,104	24	35	3	2	64	95	153	163	362	278	631	594
1897	1,130	49	52	3	3	18	21	184	176	471	263	533	490
1898	1,125	49	82	394	404	125	193	180	209	509	369	675	610
1899	1,118	27	45	100	120	257	344	115	136	354	262	481	424
1900	1,110	11	11	40	30	70	98	96	106	308	214	465	371
Mean (6–10)	1,117	32	45	108	112	107	150	146	158	401	277	557	498
1901	1,102	3	11	32	41	127	191	108	126	265	173	433	369
1902	1,099	23	31	103	113	124	164	80	81	280	184	454	384
1903	1,092	42	58	21	25	113	142	104	103	332	253	463	384
1904	1,090	25	25	9	5	77	96	134	129	192	147	268	212
1905	1,091	12	10	31	36	10	12	78	84	190	121	242	183
Mean (1–5)	1,095	21	27	39	44	90	121	101	105	252	176	372	306
1906	1,092	14	17	8	13	60	87	77	92	202	159	288	207
1907	1,089	9	9	36	36	239	283	89	107	163	109	888	802
1908	1,083	14	16	30	29	51	69	102	99	214	134	1,089	934
1909	1,080	12	23	7	8	51	70	102	102	183	124	924	830
1910	1,078	12	15	30	28	108	113	128	130	189	111	1,003	897
Mean (6–10)	1,084	12	16	22	23	102	124	100	106	190	127	838	734

Some of the data given in the above table are shown on Graphs Nos. 8 and 9, viz.:—Whooping cough No. 8 B, and typhoid No. 9 B. The results for persons for typhoid and for whooping-cough are shown on No. 9 A.

3. Secular Fluctuations of Death-rates for both Sexes together.

It will be seen hereafter that the relative numbers of males and females show no sign of progressive change, except in the cases of whooping cough and typhoid. For this reason the death-rates for persons have been prepared for the series of diseases, excepting, however, the two mentioned. The following table gives the rates per million :—

COMMONWEALTH OF AUSTRALIA.*

Death-rates from Measles, Scarlet Fever, Diphtheria and Croup, and Diarrhœa and Dysentery, 1881 *to* 1910.

Persons.

Year.	Scarlet Fever.	Measles.	Diphtheria and Croup.	Diarrhœa and Dysentery.
	Death-rate per million of population.	Death-rate per million of population.	Death-rate per million of population.	Death-rate per million of population.
1881	78	86	384	1,222
1882	70	29	387	1,409
1883	53	52	452	1,269
1884	116	168	416	1,343
1885	57	41	520	1,139
1886	45	11	380	1,065
1887	32	47	399	946
1888	52	87	496	887
1889	36	11	563	1,082
1890	65	2	724	662
1891	31	4	514	671
1892	32	1	385	619
1893	64	385	341	659
1894	64	99	307	524
1895	30	2	194	518
1896	29	2	158	613
1897	50	3	180	513
1898	64	399	221	645
1899	35	109	125	454
1900	11	35	101	420
1901	6	36	117	403
1902	27	108	80	420
1903	50	23	104	425
1904	25	7	131	241
1905	11	33	81	214
1906	15	10	84	249
1907	9	36	98	847†
1908	15	30	101	1,015†
1909	17	7	102	879†
1910	11	28	127	937†

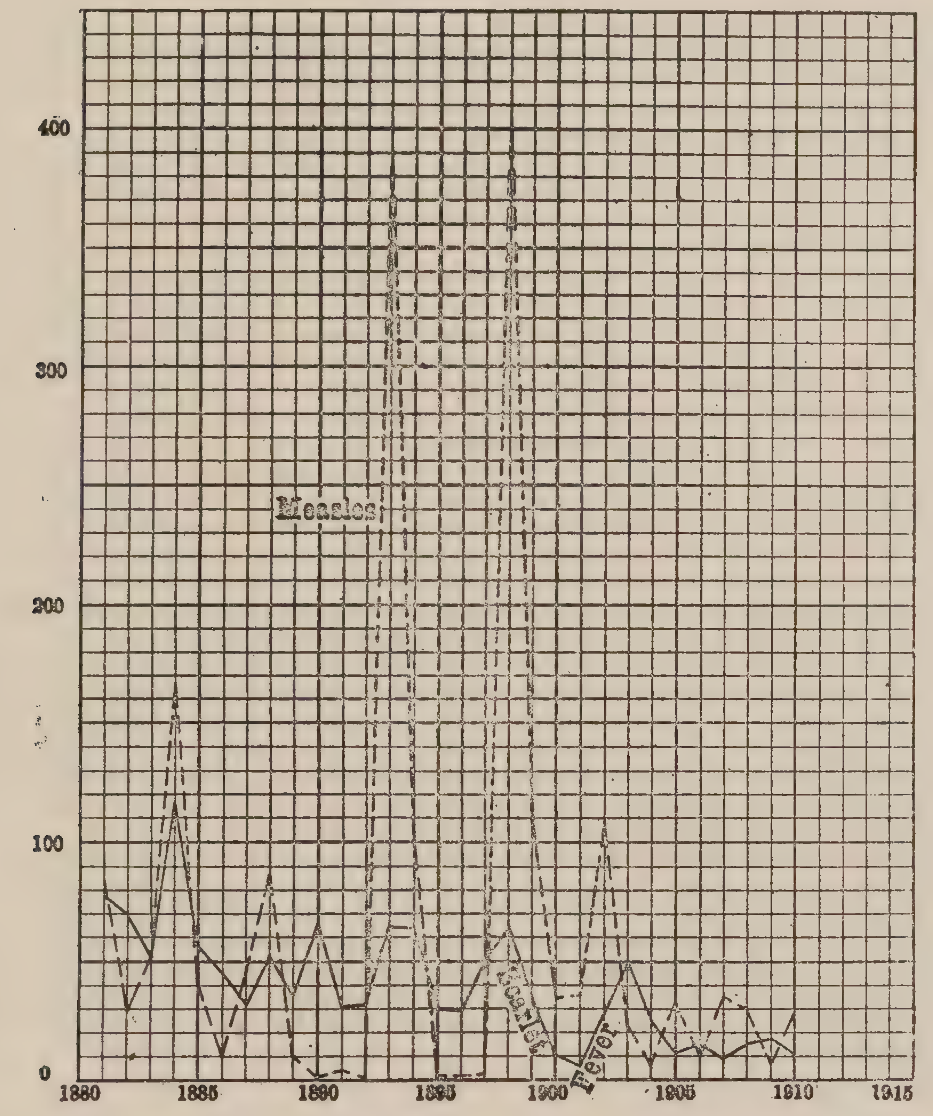
* Exclusive of Western Australia until 1897.

† Not comparable with previous years owing to inclusion of enteritis in these later years.

The data of the above table are represented in Graphs Nos. 8 and 10, viz. :—Diarrhœa, &c., and diphtheria and croup, No. 8 A; and measles and scarlet fever, No. 10.

COMMONWEALTH OF AUSTRALIA.—DEATH-RATES FOR MEASLES AND SCARLET FEVER
1881 TO 1910

Graph No. 10.



Explanation of Graph.—The base of each small square represents one calendar year, and the vertical height a death-rate of 10 persons per 1,000,000 of population. The continuous line relates to scarlet fever, and the broken line to measles. The figures on the left margin represent number of deaths per 1,000,000 of population; those on the lower margin calendar years. The diagram shows very strikingly the intensity of the epidemics of measles for the years 1884, 1893, and 1898.

4. Masculinity of Deaths by Various Diseases.

The mean quinquennial results of the previous table furnish information as to the relative frequency of death through particular diseases in the two sexes. The number of deaths of males per 1,000 females is shown in the following table :—

Frequency of Deaths of Males from various Diseases, per 1,000 *Females, when the numbers of each sex are equal, Australia,* 1881–1910.

Period.	Scarlet Fever.	Measles.	Whooping Cough.	Diphtheria and Croup.	Typhoid.	Diarrhœa and Enteritis, and Dysentery.
1881–1885	808	868	636	913	966	1,143
1886–1890	761	866	625	921	1,121	1,034
1891–1895	750	992	704	866	1,248	1,126
1896–1900	711	970	711	921	1,446	1,119
1901–1905	778	891	745	964	1,430	1,214
1906–1910	762	982	818	939	1,493	1,142
Mean of quinquennial values	762	928	706	921	1,284	1,130

It will be seen from the above table that there is no very definite evidence of any change in the relative frequency of death for the several diseases in the two sexes, except in the case of whooping-cough and typhoid. Hence the mean of the quinquennial means may be taken as the constant probability of the death of males, the probability of the death of females being taken as 1,000.

In the case of whooping-cough and typhoid this probability is a mean one for the period 1881 to 1910, but is at the present time too small, and appears to change nearly linearly with the lapse of time. Hence the masculinity in these cases must be expressed by a formula. Assuming that the assumption of a linear change represents the results with sufficient accuracy, we have for—

Whooping-cough ... $M = 593 + 7{\cdot}3\,(T - 1880)$
Typhoid ... ... $M = 951 + 21{\cdot}5\,(T - 1880)$

in which M is the masculinity, or number of males dying per 1,000 females dying, when the number of the sexes is equal.

5. Frequency of Deaths according to Sex and Age—Diarrhœa, Dysentery, Enteritis.

The following table, based on the Australian experience for the years 1907 to 1910, inclusive, indicates the distribution of deaths from these causes according to age and sex. The figures given represent the number of deaths per annum in a total population of 10,000,000, distributed according to sex and age in the same manner as the Australian population. The number of deaths from these causes is highest under 12 months, and rapidly diminishes to a minimum at about age 13, increasing thence forward to about age 77, and subsequently declining with the diminishing numbers exposed to risk.

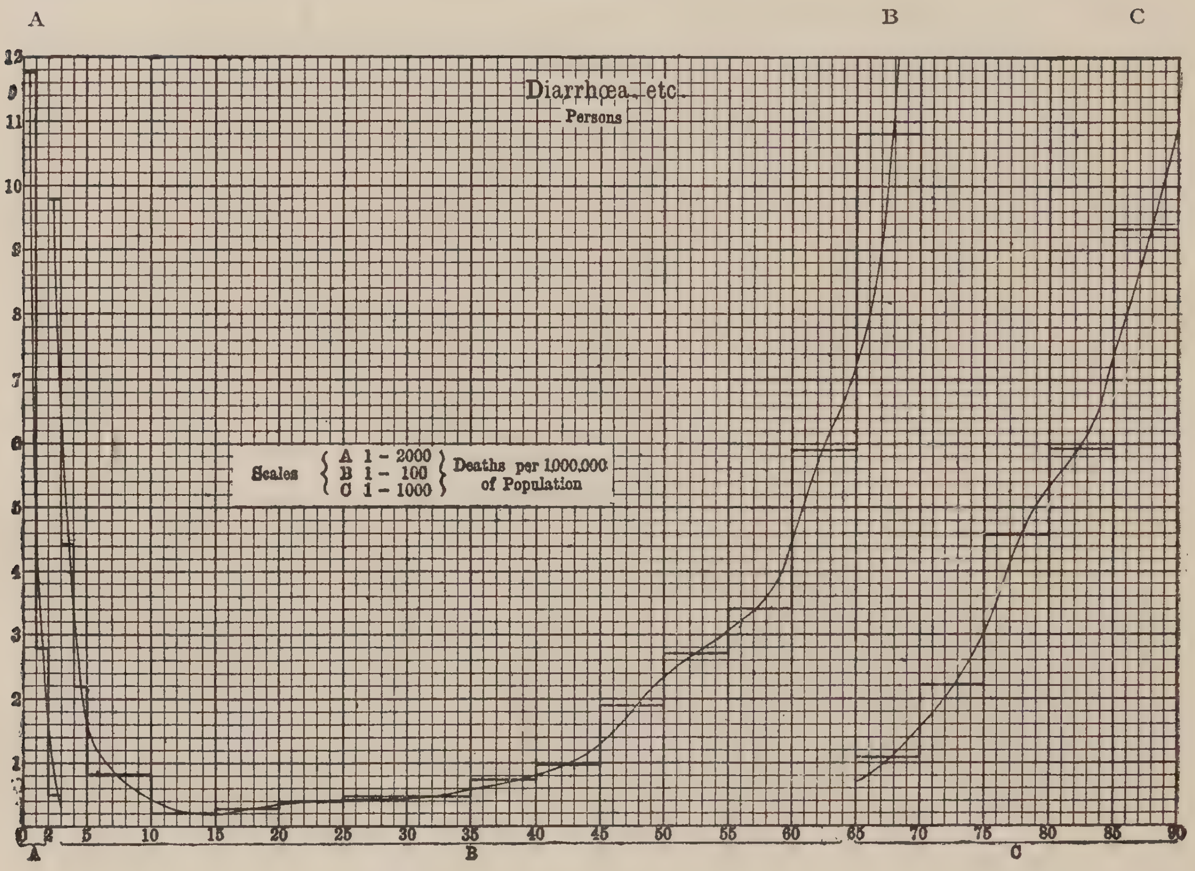
Frequency of Deaths according to sex and age—Diarrhœa, Dysentery, Enteritis. Number of Deaths per annum per 10,000,000 *of total population.*

Age-groups.	Males.	Females.	Persons.
0	3,239	2,579	5,818
1	663	582	1,245
2	102	115	217
3	52	49	101
4	25	25	50
5–9	47	54	101
10–14	15	10	25
15–19	17	11	28
20–24	15	22	37
25–29	20	21	41
30–34	19	18	37
35–39	32	27	59
40–44	31	25	56
45–49	40	38	78
50–54	52	34	86
55–59	55	32	87
60–64	67	64	131
65–69	102	96	198
70–74	144	117	261
75–79	150	120	270
80–84	99	76	175
85–89	43	37	80
90–94	12	11	23
95–99	2	3	5
100 and over	...	...	...
Not stated	3	2	5
Total	5,046	4,168	9,214

The results in this table are shown on Graph No. 3 A, in a previous paper.

COMMONWEALTH OF AUSTRALIA.—DEATH-RATES FROM DIARRHŒA, &c., ACCORDING TO AGE-GROUPS 1907 TO 1910.

Graph No. 11.



Explanation of Graph.—The curves show the death-rate per annum from diarrhœa, &c., per 1,000,000 persons of the corresponding age-group. The base of each small square represents one year of age, and the height of each small square represents 400 persons on curve A, 20 persons on curve B, and 200 persons on curve C.

6. Death-rates from Diarrhœa, &c., according to Age-groups.

The figures given in preceding section, while indicating the relative frequency with which deaths occur from the causes specified in a given population, do not give a true indication of the force with which these causes act at the several ages, since account is not taken of the respective numbers subject to risk. To present this aspect of the question, the following table has been compiled showing the number of deaths per annum in each sex and age-group per 1,000,000 of population in such group.

This brings out clearly the manner in which the rate of death from these causes diminishes rapidly from the earliest age, at which it is very heavy, to about age 13, and then increases continuously to the oldest age. The progression in rate is very similar for males, females, and persons. A graph showing the rate for persons is appended. (See Graph No. 11.)

Death-rates from Diarrhœa and Enteritis, and Dysentery, per million per annum of the corresponding sex and age-group, Australia, 1907–10.

Age-group.	Diarrhœa, &c.			Age-group.	Diarrhœa, &c.			Age-group.	Diarrhœa, &c.		
	M.	F.	P.		M.	F.	P.		M.	F.	P.
0	25,740	21,242	23,531	20–24	35	44	40	60–64	495	713	591
1	5,810	5,239	5,528	25–29	41	56	48	65–69	993	1,193	1,081
2	891	1,069	978	30–34	48	48	48	70–74	2,049	2,446	2,213
3	449	439	444	35–39	71	76	73	75–79	4,647	4,462	4,567
4	215	221	218	40–44	96	97	97	80–84	5,862	6,005	5,926
5–9	86	80	83	45–49	172	215	190	85–	11,775	7,565	9,332
10–14	27	16	22	50–54	270	273	271				
15–19	34	23	28	55–59	411	255	342				

7. Monthly variation in Deaths from various causes.

For the purpose of ascertaining the seasonal variation in the several causes of death dealt with in this paper, the number of deaths recorded in each month during the period of four years—1907 to 1910—have been taken into account. As, however, these recorded data relate to months of varying length, and as, further, the numbers exposed to risk in a uniformly increasing population are greater in the later than in the earlier months of the year, two adjustments have had to be made, one with the object of equalising the months and the other for the purpose of obtaining a uniform population basis. With these adjustments applied, the number of deaths per month per 10,000,000 of population was as follows:—

Monthly variation in Deaths from various causes, adjusted for equalised months and constant population. Deaths per month per 10,000,000 *of population.*

Month.	Diarrhœa, &c.	Whooping Cough.	Typhoid.	Measles.	Scarlet Fever.	Diphtheria and Croup.
January	1,444	85	200	21	7	50
February	1,293	98	217	9	9	57
March	1,137	72	241	8	10	78
April	996	74	216	12	11	108
May	659	99	140	12	17	122
June	376	92	118	10	16	133
July	293	124	56	12	11	123
August	239	139	48	22	12	98
September	262	130	47	36	13	96
October	417	105	36	36	11	63
November	861	99	80	46	8	73
December	1,246	95	133	27	11	72

The data above tabulated are represented on Graph No. 2 as follows:—Typhoid and whooping-cough, B; diarrhœa, &c., and diphtheria and croup, C; measles and scarlet fever, D.

8. Conclusion.

The main features of the statistical results deduced in this paper are as follows :—

(i) *Scarlet Fever.*—The death-rates are comparatively low throughout, female rates being slightly higher than males, and rates towards the end of the thirty years—1881–1910—much lower than near the beginning. The maximum rate for the period was in 1884, viz., 292 per 1,000,000. During the present century the highest rate was in 1903, viz., 193 per 1,000,000. The most fatal month in the last four years was May.

(ii) *Measles.*—The death-rates fluctuate very considerably, the disease being largely epidemic, though always present to some extent. The most severe outbreaks were in 1893 and 1898, with less severe ones in 1884, 1899, and 1902. The most fatal month in the last four years was November.

(iii) *Diphtheria and Croup.*—The heaviest death-rate was in 1890, viz., 724 per 1,000,000. Conditions have since been much improved, and rate for 1910 was only 127. The most fatal months are June, July, and August.

(iv) *Typhoid.*—Improved sanitary conditions have greatly reduced death-rates from typhoid during the period under review, the rates for later years being less than half the rates for earlier years. The maximum rate for the period was 665 per 1,000,000 in 1889. During the present century the highest rate has been 294 in 1903. The most fatal months for the last four years were February, March, and April.

(v) *Whooping Cough.*—The death rate from whooping-cough was much lighter in the later than in the earlier years of the period under review. Two years of exceptional severity were 1899 (298 per 1,000,000) and 1907 (260 per 1,000,000). The most fatal months in the four years—1907–10—have been July, August, and September.

(vi) *Diarrhœa, &c.*—The deaths from diarrhœa and dysentery, &c., have been much lighter in the later than in the earlier years. For the four years 1907–10, the statistics combine enteritis with diarrhœa, and are not thus fairly comparable with earlier years, for which enteritis was tabulated separately.

For these four years the most fatal months for diarrhœa, dysentery and enteritis have been December, January, February, and March, January having the heaviest death-rate.

DISCUSSION ON THE PREVENTION OF SYPHILIS.

The Prevention of Venereal Disease.

By B. Burnett Ham, M.D., M.R.C.S., D.P.H. (Camb.).

The promotion of the public health by legislation and other measures has occupied the attention of the section of Preventive Medicine of many Medical Congresses held in Australia. The problems of the "Black Plague" (the bubonic), the "White Plague" (consumption), the "Yellow Plague" (small-pox) have, from time to time, been the subjects of consideration and debate at its meetings; but perhaps it is not too much to say that of all the plagues which human flesh is heir to none call for more earnest attention than those venereal diseases—syphilis and gonorrhœa—which have been aptly termed the "Red Plague." The terrible consequences of syphilis in the individual damage it inflicted on the patient, the collective suffering it brought to the family, and the hereditary consequences it carried with it by the great mortality amongst infants, were tersely and unmistakably placed before the last session in addresses delivered by members in several sections of the Congress of 1908. These indictments were formidable enough, and though familiar to everyone in the medical profession, the results of the discussions have not, as yet, been productive of any material benefit to the community at large. The same negative result appears to have attended the debates on this subject at Congresses held in England and on the Continent. The reason is not far to seek. The subject has been so long tabooed in general conversation, in public speaking, or newspaper writings, that the cloak of prudery and false modesty effectually concealed from the public gaze a national evil long known to have resisted moral and religious influences, as well as the uniform testimony of medical history in every country of the world.

The prevention of venereal disease has been a vexed and difficult question, a subject unpalatable and difficult of approach, in this as in other countries, for many years past. The problem of the true relation and duties of the State to venereal disease and sexual vice has been, and still is, an international one. The conflicting claims of medical science and administrative or legal necessities subject the measures which have been from time to time proposed at various International Medical Congresses to the most contradictory criticisms. Indeed, these discrepancies of opinion between English and foreign medical authorities point to the importance of subjecting the question to an even severer critical test than it has previously undergone.

The difference of opinion prevailing in reference to almost every point at issue among the supporters of active legislation on the one hand, and the opponents of it on the other, appears to be concerned with the question as to how far the prevention of sexual vice will mitigate the whole evil. However, some conflict of opinion is both necessary and desirable; necessary, because different conclusions may be drawn by different persons from similar sets of facts, and desirable because a difference of opinion often stimulates thought and leads to further advance.

There are certain permanent facts connected with venereal disease the knowledge of which is necessary to any consideration of the subject with a view to preventive measures.

The first of these facts is the universality of venereal diseases; and by venereal disease I mean gonorrhœa as well as syphilis. These diseases have existed in every country in the world, in all conditions of civil society, in ages from the earliest antiquity down to the present times. Ignorance on the subject of venereal disease has been responsible for a tremendous mortality, which cannot in the least be estimated correctly in the reports of Registrars-General. The direct mortality may not be so very great, but the deaths indirectly due to a disease such as syphilis are difficult to appreciate, inasmuch as statistics on this point are unreliable, and frequently unrecorded. In its horrible and widespread loathsomeness, syphilis may no longer compel general attention, but it nevertheless continues to exercise its fatal power silently and secretly, and therefore deadly, because of its very insidiousness. Its protean and ever-changing forms are numerous, and often elude detection. "It respects neither virtue, nor purity, nor innocence, which are alike defenceless against its indiscriminating and corrupting influence."

Authentic statistical evidence of the extent of venereal disease in its primary and more recognisable forms is difficult to secure.

An effort was made in Melbourne recently to obtain reliable data as to the prevalence of syphilis in its various forms. Syphilis was made a compulsory notifiable disease within the metropolitan area from 1st June, 1910, to 31st May, 1911, a period of twelve months. The notification was an impersonal one, and took this form. (Notification form produced.) A circular issued at the same time to medical practioners by an advisory committee formed to assist me in the investigation requested the following information:—(Circular read.) In order to arrive at as accurate an estimation as possible, each notification was accompanied by a sample of the patient's blood for test by the "Wassermann" reaction, conducted by Dr. Hiller at the University Bacteriological Laboratory. It was agreed that only positive reactions were to be counted, and in this way a fairly accurate estimation as to the prevalence of the disease in the metropolitan area was arrived at. The scheme had these advantages: Firstly, each clinical diagnosis was confirmed or otherwise on a sound scientific basis; and secondly, the danger of over-estimation or exaggeration as to the prevalence of the disease was eliminated. The full details of the scheme are, I understand, being placed before the Congress by Professor Allen, Dr. Barrett, and Dr. Hiller in their respective sections, and therefore I will only mention here the fact that of some 5,000 cases reported 1,900 gave a positive reaction, and over 400 a partial reaction. Out of 550 cases, not included above, and reported from Dr. Barrett's clinique, forty-seven gave a positive reaction, and thirty-one a partial reaction. These latter cases included all kinds of diseases, mild and grave, and whether syphilis was suspected or not; so that 14 per cent. of all cases in this clinique were syphilitic.

At the same time that this investigation was proceeding I obtained the approval of the Victorian Government to purchase some £120 worth of the preparation known as "Salvarsan," or "606." This was obtained early in this year direct from the laboratory in Germany.

As this remedy seemed, from published accounts in current medical literature, to offer a possibility of an earlier removal of the initial lesion than by means hitherto known, except incision, some 200 doses were distributed to the various general hospitals and to practitioners for use among patients who could not afford to purchase the preparation for themselves. Records were kept as to the Wassermann reaction before and after treatment, and while a definite opinion as to its ultimate and permanent curative effects must be withheld for the present, the rapidity with which the more active and external manifestations of syphilis disappeared, even after a single injection, especially the intravenous injection of "Salvarsan," mark it out, perhaps, as the most potent and effective prophylactic agency at present at our disposal. The results were not so brilliant in the para-syphilitic conditions and nerve lesions of syphilis, but the use of "Salvarsan," in our experience, was sufficiently demonstrated to justify the hope that it may prove of great value as a preventive agent against the spread of the disease. Opinions differed as to the effect of Salvarsan upon the Wassermann reaction. In some cases the reaction changed from positive to negative in a very short time. A variation in the selection of cases as regards the disease, the method of administration of the drug, and more or less prolonged and effective treatment by mercury may account, in some measure, for the difference of opinion.

The value of the Wassermann test in diagnosis, in such an investigation as was recently carried out in Melbourne, must be apparent when to the results of the blood-examination are added the facts independently obtained by the physician, the surgeon, and the pathologist. As in no stage of the disease is a positive reaction invariable, the absence of the reaction does not imply an absence of syphilis, and, as I have said before, our estimation as to the prevalence of syphilis in Melbourne is underestimated rather than exaggerated, for the reason that only positive reactions were taken count of. If we do not know what is the exact total percentage throughout the State of Victoria, we do know that at least a very large proportion of the population are not only in danger themselves, but are also a source of danger to others.

The specificity and occurrence of the Wassermann reaction in syphilis have not been tried, so far as I know, on so large a scale for public purposes as the investigation recently conducted in Melbourne; though many authorities, notably Carl Bruck, have recorded hundreds of cases of their own in which a positive reaction was high.

Reliable and trustworthy statistics as to the prevalence of syphilis in this and other countries have always been difficult to obtain, and even such figures when available have been notoriously inadequate and misleading. The general statement has often been made that the prevalence of syphilitic diseases is so manifest as hardly to admit of discussion. A Medical Committee, appointed by the British Admiralty in 1864, to inquire into the best means of diminishing venereal diseases in the army and navy, stated that the number of admissions into the military hospitals on account of venereal diseases in 1864 amounted to 291 per 1,000 of the men serving in the United Kingdom, and that the loss of service arising from these diseases was equal to that of the whole force for an entire week. In the navy the proportion was 125 per 1,000 men, the loss of service amounting to about 586 men per day. This waste of strength does not show the total amount of injury done to the public service, since various other diseases, on account of which a considerable number of men are discharged as unfit for service, have their origin in syphilis. In 1869, syphilitic diseases, as a whole, were returned for the British army; but it was not until the year 1879 that figures showing primary and secondary syphilis were shown separately.

Lieutenant-Colonel Melville, Professor of Hygiene, Royal Army Medical College, England, shows that the admissions for syphilis in the British Army by certain stations for certain periods were as follow:—

	1887–96.	1897–1906.	1903–7.
United Kingdom	95·1	41·5	31·7
Gibraltar	48·4	24·1	18·3
South Africa	134·4	19·6	23·4
Ceylon	130·1	56·1	24·9
Straits Settlements ...	235·2	72·8	40·6
India	189·7	99·0	43·0

It will be seen from this table that there has been considerable improvement in the British Army of late years, and that the majority of the stations mentioned are important naval, as well as military, stations, "a combination that in the United Kingdom has always been considered to have a detrimental effect as regards prevalence of venereal disease."

The decrease is especially notable in India, where "the number of men permanently non-effective as a result of syphilis was 1,760 in 1896, and only 162 in 1908, a decrease which is one of the most striking facts in military epidemiology, and the officers and men of the British Army in India," says Colonel Melville, "have every right to boast of a change due not to any penal restrictions on unfortunate women, but to a general raising of moral tone, and the growth of a healthy self-respect, in the production of which both have taken their share."

To take the latest figures available:—"In the year 1908 the army of the United States showed an admission rate of over 26 per 1,000; the British Army in the United Kingdom, 20 per 1,000; whilst in 1906, for the German army the figures were 4·7, the Bavarian 3·6, the Austrian 16, and the Belgian 6·12; the Italian rate for 1902 was 14·5."

The difference between these ratios is not, to quote Colonel Melville, to be attributed to any marked influence by the enforcement of repressive measures so much as to the voluntary method of recruiting in England and America, and there is no evidence to show that in the case of the civil populations of the countries concerned there is any marked difference in the prevalence of syphilis. The intense application to military training as is demanded by Germany and

France gives the recruit little leisure or money to spend. As Melville tersely puts it, " Want of opportunity is the best preventive of syphilis, and want of money does not come far behind it." The data available of the prevalence of syphilis in the British Royal Navy is less easy to obtain than in the army, and the figures are less accurate. It is stated that syphilis is still prevalent in the British Navy in comparison with the navies of other Powers, but that, with the exception of the Australian Station, where there has been a progressive increase, the ratio on all stations has undergone a reduction in the past twenty years. According to official returns, the prevalence of syphilis on the various stations, from the average ratios per 1,000, show that for primary syphilis the Home Station in 1866-1875 was 40; from 1896-1908 it was 37; for the Mediterranean Station for the same periods it was 39 and 23 respectively; for the Pacific Station 62 and 22; for the China Station 94 and 43; and for the Australian Station 29 and 39.

I have taken the service as they probably afford a basis for as accurate an estimation as it is possible to secure. Though of late years the treatment of syphilis in the British navy has attracted more attention than formerly, preventive measures of a personal nature have never been officially recognised in either the British army or the navy, in so far as the practical measures against the spread of syphilis are not enforced by disciplinary measures as now prevail in some foreign navies. In the German navy personal prophylaxis in regard to venereal disease is made a matter of discipline, and is enforced by punishment of those men who, having become infected, had not made better use of the preventive measures which were placed at their disposal.

These compulsory prophylactic measures, which I will refer to later, have been the means of effecting a marked reduction in the number of cases of venereal disease occurring in the German navy. Other foreign navies are adopting these measures, and the leading officers of our own British navy are holding out the hope that similar measures will soon prevail for the British sailor.

Of less authentic statements, as showing the prevalence of syphilis, it is stated that 50 per cent. of the enlisted men of the United States navy have a venereal disease during an average of four years. " During 1908," Dr. Fiske, of Washington, states, " there were 4,681 original venereal infections, which entailed a loss of service and expense for treatment of 106,526 sick days." " Sailors and marines," he says, " do not contract venereal disease from the navy, but from civil communities notoriously indifferent to venereal disease." Military services have practised compulsory notification of venereal disease for years, and expect civil authorities to undertake similar measures for control. With regard to civil communities, among the adult population of France the percentage of known syphilitics among the adult males, according to an authority working on hospital statistics, is 13 to 18, and yet in France there is a system of control. " The dangers of syphilis," says Havelock Ellis, in his book on the " Psychology of Sex," " lie not alone in its potency and persistence, but also in its prevalence." " It is difficult," he says, " to state the exact incidence of syphilis, but a great many partial investigations have been made in various countries, and it would appear that from 5 to 20 per cent. of the population in European countries is syphilitic, while about 15 per cent. of the syphilitic cases die from causes directly or indirectly due to the disease."

In the city of New York not less than a quarter of a million of cases of venereal disease occurred every year, while in Germany 800,000 cases of venereal disease are, by one authority, estimated to occur yearly.

Enough, then, has been said to show that venereal disease is not only one of the most appalling of all diseases, but perhaps the dominant disease of the world.

The prevalence and the potency of syphilis call loudly for plain, practical, common-sense reform. It must not be imagined, however, that the significance and prevalence of syphilis have not called forth attempts at its prevention. Preventive measures have not been delayed because of ignorance of the precise nature and causation of the disease. On the contrary, for a great number of years attempts have been made in many countries to prevent syphilis, but they have failed, not because they have been few, but because they have been inadequate. They have failed, and they always will fail, to reconcile the moral with the medical aspect of the question.

The weakness of all past and many existing efforts to control syphilis is that they are directed against prostitution as cause and effect, or that they assume that the remedies for both are likely to lie in the same direction. The problem of prostitution is not simply the problem of syphilis. "The two questions, however intimately they may be mingled, are fundamentally distinct."

The problem of prostitution would still exist, even if venereal disease could be brought under the same control as small-pox or leprosy. History proves the ineradicable nature of prostitution. There is no case on record of its ever having been completely and permanently suppressed in any community. No moral, religious, or legislative methods of treatment have been of much avail, but on the contrary "all direct attempts to suppress the evil of prostitution, whenever or wherever they have been made, have only resulted in aggravating its intensity." This fact is admitted by all writers on the subject, even by those who are the most strongly opposed to the policy of legal toleration.

Many International Congresses on the Prevention of Syphilis and Venereal Disease have been held since the one at Paris in 1867, and the one in Brussels in 1899. The latter conference was attended by 360 members, of which 107 were Government delegates from twenty-nine different countries.

The proceedings of this Conference, and of a subsequent one held in 1902, are illuminative of the fact that though the value of regulation—compulsory registration and periodical examination of prostitutes—was hotly debated, yet no resolution on the subject was ventured by either Congress. In fact, it may be said that no international conference held as yet to deal with the prevention of venereal disease has submitted conclusive and consistent evidence of the diminution of the disease by coercive measures.

The fact is, that prostitution as it now exists has become more intimately mingled with social life generally, and prostitutes are less distinguishable as a definitely separable class. Germany has a strict system of supervision by means of registration, but the numbers of non-registered prostitutes is estimated to be at least three times that of the registered. In France, the registered women in registered houses must be at least 21 years of age, and experience has shown that if girls can be tided over to that age they are less susceptible to temptation. The registered women are visited weekly by a physician, but the unregistered women who solicit in secret are considerably larger, and are mostly minors, highly susceptible to venereal diseases, and very dangerous to the public health.

Dr. Blaschko, of Berlin, in opening the discussion on the influence of existing systems of regulation on the prevalence of syphilis at the International Conference held in Brussels in 1899, said:—"The only test of the value of regulation is the frequency of venereal diseases among the female population." He had made a careful examination of all available reports and statistics, and "the result is that I can find no distinct evidence of a beneficial result from regulation, or of a harmful result where it is absent or abolished."

Professor Fournier, of Paris, said:—"Whatever the authorities have been able to do in the way of regulation, it still remains inadequate; and the disease abounds and superabounds to-day as it did before regulation existed."

A study of the voluminous reports of the International Conferences on this question makes it abundantly clear that regulation of prostitution has failed, chiefly because "it can only deal with a particular section of infected persons, leaving a much larger number at liberty to spread the disease; and secondly, because it fosters the immorality which is the ultimate cause of disease."

It is pertinent to ask here: What is a "prostitute"? The definition is important from the legal standpoint, because the exact point at which a woman begins to be a prostitute is a question of considerable importance in countries in which prostitutes are subject to legal control. In the United States prostitution is less open in some cities,—as, for example, New York—than in others; but the social evil is probably more widely spread through all classes, from the very wealthy to the very poor. In some cities like Boston, there is, perhaps, less open prostitution, though a fashionable avenue is almost lined with the apartments of kept mistresses. Even in Japan, where prostitution is organised like the postal or telegraph service, there is much clandestine prostitution. But we have still to define the term "prostitute," for it is essential that in any State regulation of vice, a more or less sharp line of demarcation must be drawn between the class recognised by law as prostitutes and a class, certainly larger and certainly more dangerous, which the law cannot or will not touch.

From the medical point of view, with which we are directly concerned, and from the moral point of view, with which we cannot fail to be indirectly concerned, adherence to any rigid definition of what constitutes a prostitute must necessarily limit the number of registered women to a point wholly incompatible with even a plausible show of medical efficiency. If one fact more than any other stands clear in all the reports of international conferences, medical and otherwise, on this question, it is that in no country does the administration succeed in enrolling all the women, whilst the offered safeguard is illusory, inasmuch as the most efficient and carefully conducted examination can afford no guarantee of safety. The more strict the supervision over registered women the more numerous the "clandestine" class, and the more risk of syphilitic infection among the latter.

Fournier says, "We are helpless to deal with the fashionable courtesan." But the problem is not so simple as has been stated by Fournier. "You ask me," he says, "if I am content with regulation. No, I am not; yet I am in favour of it because it is better than nothing at all. . . . It controls but a small number of women, but at least it controls them."

But it is not the question of a woman, but women, and the class of women not necessarily that class designated as prostitutes. Fournier himself has shown that minors and novices are the most dangerous as regards syphilitic infection, and the most apt to elude police supervision.

Nor is it simply the question of the woman, but of the man as well. Professor Gaucher, who has since succeeded to Fournier's post at the Hospital St. Louis, Paris, says:—"In regard to the prophylaxis of venereal maladies, recourse must be had to common law, equal for men and women."

From the medical and hygienic points of view, no one will contend that man's vice is to be treated as a fixed quantity, and no interference with it of any kind to be resorted to. Promiscuous indulgence on the part of a man suffering from venereal disease is no less derogatory and discreditable to the man who can afford to pay for his vice than to the woman who is poor enough to be tempted to accept payment, and infection, from him.

The main and primary object of any State regulation of vice is the restriction, the mitigation, and the cure of venereal disease which are traceable to the promiscuous intercourse of men and women. The regulation and licensing system as practised on the Continent therefore must fail, from the medical point of view, if the absolute sequestration of all diseased women and men cannot be secured. Even if it were expedient to regulate prostitution by more effective and less harsh measures, the method of dealing with this evil must necessarily change, seeing that the character of prostitution as practised to-day has also changed. The old-time brothel and the official system of regulating the same are alike out of date, and the repugnance to brothels in many essential respects also applies to prostitution generally.

On the whole, it may be said that the system of registering, examining, and regulating prostitutes belongs to the past. "The objection to the regulation of prostitution is steadily developing everywhere, and may be traced equally in scientific opinion and in popular feeling."

A consideration of the system of licensed prostitution abroad would not be complete without some brief reference to the form it took in the passing by the English Legislature of the Contagious Diseases Acts.

"There are no doubt some characteristic differences," says Sheldon Amos, "in the mode of applying the system of licensed prostitution long adopted on the Continent and the mode introduced into England. These differences are due partly to the peculiarities of the English Constitution to which the new system had to be made at least plausibly conformable; partly to the religious and moral objections which a system could not but encounter in England in proportion as the existence of it became divulged, and the nature of it understood; partly to the lengthened experience gained in other countries, which the founders of the system in England were able to avail themselves of in their attempt to create a structure which might at least ostensibly seem proof against all the assaults on moral and constitutional grounds to which the system, as it has hitherto existed abroad, has been intermittently exposed."

The main object of the method abroad was to protect the national military forces, rather than the civil population, from the terrible physical consequences accruing from indulgence in the vice. In countries maintaining large and costly standing armies, the legislative enactments dealing with the matter might fitly have been termed "military sanitary regulations." In England the system was enforced by statute, and in the express terms of a statute the powers of the police, the duties of the surgeons, and the amounts and qualities of the penalties described, and, so far as possible, strictly limited. "It is necessary to insert the words 'as far as possible,' because it was clear from the nature of the case that unless very large and indefinite powers were conceded to the police, such as are conceded to them for no other purpose whatever, even a plausible prospect of sanitary success was unattainable."

While the intelligence and humanity of the police generally may or may not be called into question, "the degree in which these qualities are exhibited depends on the accidental temperament of the particular officer or on the casual view he chances to take of the nature of his responsibilities." Up to the passing of the Contagious Diseases Act in 1864, there was in England no direct registration dealing directly with prostitution as a means of controlling syphilis. "The first of these Acts was avowedly tentative in its character, and its operation was restricted to a few large garrison and seaport towns." The main provisions of this Act were that if an inspector of police or any registered medical practitioner had good reason to believe that a certain woman was a common prostitute, and had a contagious disease, the justice might call upon that woman to appear to answer the charge, and upon substantiation of the charge the woman was to be taken into a certified hospital for medical examination, and, if found diseased, she was to be detained for a period not exceeding three months. The places to which the Act was made applicable were certain military and naval stations such as Portsmouth, Woolwich, Cork, and so on. The adoption of this Act had been forced upon the British Government by the prevalence of venereal disease in the army and navy. The bill received the Royal assent in July, 1864. In the October following the Government appointed a committee of eminent medical men to investigate its working. The committee reported, after an exhaustive inquiry occupying more than two years, that "in one most important point the Act had proved successful, and in just that particular in which it might *a priori* have been expected to fail, namely, that which related to the feelings of the unfortunate women with whom it had to deal;

so far from opposing its operation, they appear to appreciate its value to themselves." The defects of the Act were mainly stated in the fact that it did not enable the authorities to lay their hands on the disease and eradicate it in its earliest stages, when it is most susceptible of cure. The committee recommended amendment of the Act in many of its main provisions and its extension to other localities; a recommendation which the Government carried into effect by introducing and passing the Amendment Act of 1866. The amended Act was read a second time in the House of Commons in March, 1866, when a brief debate took place, the first that had been raised on the subject in Parliament. Mr. Henley characterised the Bill as a "very queer Bill on a very queer subject, and an endeavour to remove all the penalties which a Higher Power had imposed upon sin, and to give the opportunity of sinning without the punishment." One of the main provisions of the amended Act was to the effect that where an information on oath was laid before a justice by a superintendent of police that a woman was a common prostitute, the mere fact that such a woman was a common prostitute was sufficient to justify her examination, even though she was not suspected of being diseased.

Provision was also made for the establishment of certified hospitals. A Select Committee of the House of Lords was appointed in 1868 to consider the Act of 1866. This committee reported that defects in the working of the Act of 1866 had arisen mainly from its limited scope, and the consequent intrusion of unregistered and infected women into the scheduled districts, and they recommended the cautious extension of the Act to all the naval and military stations. A fresh Act was passed in 1869, and a Select Committee of the House of Commons was appointed to inquire into the working of the 1866 Act. This committee reported that during the two and a half years the Act had been in operation much benefit had accrued, and that prostitution appeared to have diminished, its worst features to have been softened, and its physical evils abated.

It is a significant fact that in all these Acts little or no discussion took place in either House of Parliament, the Government accepting and acting upon the recommendation of the various committees appointed; but on the 24th May, 1870, Mr. W. Fowler moved, in the House of Commons, for leave to bring in a bill to repeal the Acts of 1866 and 1869. A long debate ensued. One petition, signed by 270,000 persons, and another with 500,000 signatures, were presented to the House, condemning the Acts. The Government again shirked the responsibility of dealing with the matter, and appointed a Royal Commission of Inquiry. This Commission consisted of twenty-five members, of whom twenty-three signed the report, which was presented to the Queen in December, 1871. The commissioners were much divided in opinion. Two-thirds of them were in favour of a qualified system of compulsory registration, seven were in favour of strengthening the terms of the Act, and six in favour of repealing all compulsory legislation. All were in favour of further legislation, with a view to modifying the law as to make it applicable to the whole country. The Commission, among other things, recommended that the periodical examination of public prostitutes be discontinued. This recommendation is somewhat difficult to follow logically, for if women are to be submitted to enforced examination at all, then surely it is better to make such examination a periodical affair and a matter of ordinary routine. Either examination is right or it is wrong. "If it is right, then it should be made as wide-spreading as possible; if it is wrong, it should be stopped altogether." The growing opposition bore the usual fruit in the shape of a Select Committee of the House of Commons, appointed in June, 1879. The report of this committee dealt with the hygienic, constitutional, moral, and social effects of the Acts. Briefly summarised, they found that the Acts had diminished, to an extent, syphilis, and, to a less extent, gonorrhœa, in the subjected districts; that the chief objections urged against the continuance of the Acts were:—(1) That they violated the first principles of constitutional law; (2) that they involved the State in recognition of vice; (3) that they subjected women to restraint from which men were free; (4) that they increased vice by affording immunity; (5) that they subjected women to the risk of police persecution; and (6) that they increased clandestine prostitution.

On all points the members of the Committee were at variance. Some considered that the constitutional position was the same as in the case of the law enforcing vaccination; that the State did not recognise prostitution to any greater extent than the fact that prostitutes were openly permitted to ply their trade; and that the Acts were not aimed at women, but at a class of women only.

The divided character of the Commission, and the persistent agitation against the Act by the general public, induced the Government to bring in a bill in April, 1872, repealing the Acts of 1866 and 1869, and substituting for them an entirely different system. Under this new bill it was provided that "the act of importuning persons in the streets should bring a woman under its operation, even though the importunity does not cause annoyance or create disorder, and though it be not proved by the evidence of the persons importuned. Any common woman so soliciting in a public street was rendered liable to imprisonment, and if during her imprisonment she was found to be suffering from a contagious disorder she could be removed to a certified hospital, and detained there for nine months or until cured. The bill also gave power to prosecute the keepers of disorderly houses, and keepers of common lodging-houses harbouring prostitutes were similarly made liable.

The new bill met the opposition both of the Parliament and the Press, and it was withdrawn by the Government.

A memorial to the Home Secretary, signed by many hundreds of medical men in the United Kingdom, was presented in 1872 in favour of the Acts. On the other hand, a powerful opposition to the Acts in the shape of 873 petitions, with 622,258 signatures, and another petition signed by a quarter of a million of the women of the United Kingdom, were presented or ready for presentation to the Prime Minister.

Within nine months of the issue of the report by this last Select Committee of the House of Commons, a recommendation of the minority as to the abolition of the compulsory examination of women was adopted by an Order, and in 1866 complete repeal followed by the passing of the Contagious Diseases Repeal Act. Since that date there has been no revival of the system in England.

I have sketched the history of the English Contagious Diseases Acts at some little length, because in several of the States of the Commonwealth attempts to carry similar legislation into effect have been made from time to time. An adaptation of the English Act was introduced into Parliament by the Government of Victoria in September, 1878, under the title "A Bill for the Prevention of Contagious Disease." It passed its second reading in the Assembly, and a new title was proposed on its third reading, viz., "A Bill for the Conservation of Public Health." It reached the Legislative Council in November, 1878, under this title, but was withdrawn after its first reading.

The provisions of this bill were subsequently incorporated in the Public Health Act of 1890, which is the health statute of the State to-day; but, owing to the opposition of many influential religious bodies, the provisions of Part VIII of the Health Act, under the heading of "Syphilis," have never been enforced by any Government, and they are inoperative at the present time.

A similar bill was introduced into the South Australian Parliament at an earlier date than the Victorian bill. It passed the Legislative Assembly, but was rejected at the third reading by the Legislative Council.

Similar legislation was introduced in 1876 into the Legislature of New South Wales, and was a bill closely resembling the English Contagious Diseases Acts of 1866 and 1869, as construed together. A suggested new clause to the bill of 1876 was that women were required to name the persons suspected to have imparted contagion, and upon the name of the male being disclosed such male person was required to attend for examination by the visiting surgeon. This bill met a similar fate to other suggested colonial legislation.

A Contagious Diseases Act, providing for the licensing and medical examination of prostitutes, has been in force in Brisbane for many years. Syphilis and gonorrhœa are common diseases in Brisbane, both in their primary and secondary manifestations. In a paper contributed by Dr. A. Jeffris Turner at the last Australasian Medical Congress in 1908, Dr. Turner stated:—"With the whole of the male sex, and the most dangerous carriers of syphilis in the female sex, excluded from its operations, it is not surprising that the Act accomplishes little in the prevention of disease. . . . The result has been a miserable compromise, neither free from moral objection on the one hand, nor of any appreciable value in preventing disease on the other."

It would appear, then, that in any country the periodical examination of all suspected persons is impossible. Women, or a certain number of them, can be got hold of; but men cannot be drawn into the net of the Contagious Diseases Act. It is contended by the supporters of such legislation that at least a few women can be controlled, and that that is better than nothing at all. The argument would be sounder if the alleged necessity of this particular vice, and the proposed State action, could claim a moral apology for exceptional and anomalous legislation. Nor does it find a parallel in other vices calling for State control. "It may be," says Colonel Melville, in his article on "The History and Epidemiology of Syphilis in the more Important Armies" (in "A System of

Syphilis"), "and is, quite justifiable for the State to recognise the drink trade, and to regulate it so as to minimise drunkenness, since the latter is by no means a necessary corollary of the former. But the regulation of prostitution to prevent venereal diseases is a different matter. Drunkenness is the result of drinking to excess. Venereal disease is not the result of excessive impurity. Looked at from the purely social point of view, drinking is no sin, whereas drunkenness is. On the other hand, fornication is a social sin, but venereal disease a mere misfortune. It is morally quite justifiable to so regulate an essentially innocent act as to prevent the excess which makes it a social crime, but it is not justifiable to regulate the practice of a sin in order to save those who indulge in it from the possible consequences."

As the legal duties of persons are very seldom coextensive with their moral duties, the State, which is merely the sum of its individual members, can go a very little way indeed in directly promoting the morality and virtue of its citizens. "How far the State can wisely attempt to go is a matter upon which the ablest thinkers and practical statesmen are by no means at one." "Weak laws, by proclaiming their own impotence, can only add to the evil they affect to remedy."

The medical theory on which Contagious Diseases Acts and the licensing system rests is that of their value as purely sanitary agents. It is on sanitary grounds that this system has been introduced, and it is on sanitary grounds that it must be mainly defended. If it cannot be defended on these grounds it can hardly be upheld for any other reasons. The purpose of the whole system was originally and essentially sanitary. The method applied is that of bringing under medical observation and treatment all women capable of communicating the disease.

The criterion for admission to the class and retention within it is her liability to communicate disease. From the moral point of view all doubtful cases must be excluded. From the medical point of view all doubtful cases should be included.

The directly moral bearing of the system under consideration I will not attempt to discuss. The business of the sanitarian is to prevent disease, and not to preach morals. It is, as said before, difficult, if not impossible, to reconcile the medical and moral aspects. They are incompatible.

More than thirty years ago a majority of the medical profession were in favour of the regulation system. To-day medical, military, legal, ecclesiastical, educational, and sanitary authorities combine in condemning it with a distinctness of utterance and plainness of speech that cannot be misunderstood.

In 1864 the Contagious Diseases Act was forced upon the English Government by the prevalence of venereal disease in the army and navy. To-day, the English Army Medical Advisory Board appointed to inquire into the prevention of venereal disease in the army states:—"The remarkable diminution of venereal disease among British troops in the stations where the Contagious Diseases Acts were in force is an example of evidence which, at first sight, appears to bear the most cogent character. . . . It is noteworthy that in certain foreign countries where the police supervision of prostitution has been carried on much more strictly than was ever attempted in the United Kingdom serious doubts are now expressed as to the efficacy of legal restraint in arresting these contagious diseases. . . . Taking into consideration the present state of expert opinion abroad, and the opposition certain to be raised if a statute on the lines of the Contagious Diseases Act be proposed, the Committee has come to the conclusion that, in the United Kingdom at any rate, an attempt to grapple with the problem of venereal disease by methods of compulsory isolation and treatment is neither practicable nor expedient."

The study of this system for the prevention of venereal disease leads us, therefore, to the conclusion that not only in the United Kingdom, but on the Continent, such measures have been condemned of late years, for the reasons that they are not only unjust, but because they are inadequate and of very doubtful utility.

But we ask ourselves the question: Since such preventive measures have been tried and have been found wanting, is nothing, then, to be done?

Much has already been done, and much remains to be done. As showing the direction in which sanitary science is moving in a variety of places widely separated in their geographical distribution and their political institutions, some very brief account of the methods of combating venereal disease in these countries may not be out of place were it not that I am fully cognisant of the fact that my remarks have already detained you quite long enough, and I have yet to place before you the "plan of campaign" a study of this subject invites. Therefore, I will be as brief as possible.

In Germany, a law exists making it compulsory on every civilian doctor treating a soldier for venereal disease to report the same to the military authorities.

In the French army, a circular is issued enjoining on medical officers the duty of warning the men of the dangers of immorality, and of the necessity of early treatment.

In the German army, it is obligatory on every soldier suffering from venereal disease to seek treatment, and power exists to enforce such treatment if necessary.

While the young soldier is thus taught that not only is continence possible and healthful, and the dangers of immorality pointed out, yet cleanliness is the first lesson insisted upon.

The use of a protargol-glycerine and a calomel ointment is recommended, and made available to the men without payment. Infected soldiers are punished.

In the Army of the United States, personal prophylaxis on the above lines has also been practised, especially in the Philippines, where venereal disease appears to be prevalent. The "venereal prophylactic kit" consists of a small pasteboard box containing a solution of argyrol and calomel ointment. Brief directions are placed in each box, together with a plain written pamphlet warning the soldiers against the dangers of the disease. An extension of these prophylactic measures to civil populations has become a natural and necessary step.

In many countries at the present time the problem is resolving itself into one of education.

In England, in Germany, in France, in America, in Denmark, in Norway, and in other countries societies are being formed for the prevention of venereal disease. In Germany, one society already numbers some 5,000 members, including eminent medical men and ladies of high social position. This society has alone distributed a million and a half copies of pamphlets to the public.

A society, known as the "Red Plague" Society, in California, is doing excellent work by means of lectures, pamphlets, and addresses. The idea is to secure that moral stability among young men and women which is the greatest personal measure of safety.

"The fear of syphilis and gonorrhœa is the beginning of wisdom." One of the most important resolutions passed by the Second International Congress on Syphilis at Berlin read:—"The public must be taught that, instead of being ashamed of these diseases, and not fearing them, it must not be ashamed of, but fear them." Again, in 1902, one of the resolutions of the Brussels Conference passed ran:—"The most important and the most effectual means for combating the diffusion of venereal maladies consists in widespread information as to the importance of these maladies, and the very grave dangers attending them. It is especially necessary to teach young men not only that chastity and continence are not injurious, but that these virtues are highly recommended from the medical point of view."

Ignorance is, to a great extent, the root of the evil. Knowledge is needed at every step to guide and awaken the greatest safeguard of all, the sense of personal moral responsibility—"self-knowledge, self-reverence, self-control."

We must drag these diseases into the glare of public opinion. We must fight them freely, but openly. Vague declamation from the pulpit, and still vaguer writing in the Press, should give place to a dignified, simple, and concise statement of the actual facts. That the general public is beginning to realise the malign influence of these diseases is proven by the fact that the London *Times* recently published an article on syphilis, calling it "Syphilis," and not "a specific disease," "a certain dangerous disease," or other vague appellation. In Melbourne the National Council of Women, the religious bodies, and friendly societies, are beginning to take the matter up, and the Press has already published certain articles on the recent investigation, and calling the disease by its proper name. The youth of our country has been taught for many years through the advertising columns of the public Press that these diseases are easily avoided or cured by means of quack remedies and quack treatment. There is thus the responsibility of the Press. Then, again, there is the responsibility of parents to cast off the old, unwise attitude of secretiveness and repression of the knowledge of sex matters.

Some people have the idea that these diseases are only contracted by the lewd, and that they form a really salutary punishment for sin. The fact that the innocent and just, that children and babies, may also suffer is apparently lost sight of in the outbreak of indignant but misplaced protest on the part of these moral enthusiasts. We must also give up the idea that the syphilitic is an outcast, a leper, or a social and moral derelict. So long as human nature remains what it is, and it is much the same to-day as it was centuries ago, it is useless to preach morality to young men and women endowed with strong animal, but not the less natural, passions. Such counsel of perfection is very much like the advice of the mother to the child desirous of learning to swim, "Hang your clothes on the gooseberry-bush, but do not go near the water."

There is also the responsibility of the medical man; and here, perhaps, there is a deeper responsibility than even upon the parent. We, at least, cannot plead ignorance of the nature and effects of syphilis and gonorrhœa.

Of equal importance to instruction and education on these diseases is the provision for efficient treatment for their victims. It will hardly be maintained that the treatment of the majority of cases of venereal disease in the out-patient departments of our general hospitals is satisfactory or adequate. Regular and prolonged attendance of patients is difficult to secure, and the patient no sooner finds himself free of the more active manifestation of his disease than he imagines his troubles over, and that he is at liberty to throw discretion to the winds.

In the army it has been found that in spite of advice and warning the soldier will persist in running the risk. A rational method of prevention is placed at his disposal, and if he neglects to make use of it he breaks the regulation, and suffers in consequence.

At Fort Benjamin Harrison, in the United States, men who wished could make use of an ointment consisting of 25 per cent. calomel. This was furnished in a collapsible tube, with a cone-shaped point. In a series of 1,200 possible exposures no case of infection resulted.

The maintenance of free public hospitals for the isolation and cure of venereal disease has apparently met with less prejudice than the admission of venereal patients to the wards of general hospitals.

Without entering into the cause which actuates hospital authorities in refusing admission to the unfortunate sufferers of syphilis—guilty or innocent—one fact is clear, that with regard to "special" or "certified" hospitals under the regulation system, the applicant must first qualify as a registered prostitute.

Hospital treatment, then, instead of being a redeeming feature of the problem, is rather an aggravation of it. As a general principle, all disease, however occasioned, has a presumable claim to equal hospital treatment.

The public recognition of a public or general hospital is that it is an establishment which should relieve, without question, all persons of both sexes suffering from disease, merely because it is suffering. No hospital board or committee can constitute itself a moral tribunal and attempt to proportion its treatment or withhold it altogether from the innocent and guilty alike because, in the long run, the guilty are likely to be the most frequent applicants. To appoint any general hospital (or special ward therein) receiving aid from the State, a place in which persons of both sexes suffering from venereal disease may be placed and detained for medical treatment, cannot either promote immorality or infringe personal liberty to any greater extent than that the moral duty is made, as far as possible, coextensive with the legal necessity. It would at least be free of the stigma attaching to a special or lock hospital.

A method of venereal prophylaxis that recommends itself seems to be one which has been found useful in some countries, and notably in Denmark, which country has adopted the Scandinavian plan in a modified form. This little country for some time followed the example of her great neighbour, Germany, by adopting the police registration of venereal disease and prostitution; but since the year 1906 Denmark has practically led the world in its management of venereal disease. The system of regulation was entirely abandoned, and Denmark resolved to rely upon thorough and systematic application of the sanitary principle of affording free treatment for any of its inhabitants. Compulsory notification is exacted, the privacy of the patient being safeguarded by a number. Everyone, whatever his or her social or financial position, is entitled to free treatment of venereal disease. Whether they avail themselves of it or not, they are in any case bound to undergo medical treatment. In this way every diseased person is, so far as it can be achieved, in a doctor's hands. All patients are warned by the medical men as to the danger of infecting others, and as to the legal penalty attaching thereto. Neglect on the patient's part calls for police interference, compulsory treatment, and segregation. It is largely due to the systematic introduction of facilities for gratuitous treatment that the enormous reduction in venereal disease in Sweden, Norway, and Bosnia is attributed.

With regard to the transmission of venereal disease, legal cognisance has been taken in several countries. In England, the communication of venereal disease is not an actionable wrong if the act of intercourse has been voluntary.

In the French courts, heavy damages may be obtained from men who have infected young women. In Germany, efforts are being made at the present time to declare by law that sexual intercourse on the part of the person who knows that he or she is diseased be punishable by imprisonment for a period not exceeding two years.

"The only grounds on which venereal disease could deserve to be selected out of all diseases for gratuitous care and treatment, or punishment of offenders knowingly disseminating them, must be that they exceed in danger to the community or suffering to the patient every other disease."

Of the medical facts there can be no dispute. Of the legal principle involved, just and wisely-adopted legislation should claim equal allegiance on the part of the man as well as woman, and the penal offence for voluntary communication be the moral and logical justification of an adequate curative and palliative treatment gratuitously supplied and previously offered. But, in any case, the preventive and protective aspects must far exceed in importance the strictly curative aspect of the question.

I am well aware that a number of other and difficult questions are still left open for discussion, but it now only remains for me, in conclusion, to place before you for your consideration the following table of conclusions to which the preceding review of the subject seems to point.

I. Legal and Administrative.

(I) *Compulsory Notification of Syphilis and Gonorrhœa.*
  (*a*) Notification by number, initial, or letter.
  (*b*) In cities and larger towns only.
  (*c*) Form of notification.
  (*d*) Free bacteriological examination and "Wassermann test."

(II) *Prostitution* to be dealt with solely as a matter of law and order; immoral literature, the decency of the streets, solicitation, and juvenile vice to be dealt with under Police Offences Acts and similar Statutes in each State.

(III) *Compulsory and Gratuitous Treatment.*
  (*a*) All persons suffering from venereal disease to be medically treated either at a general hospital, or by private medical practitioner, or medical officers appointed by Government.
  (*b*) Treatment to be compulsory by medical profession only, easy of access, adequately supervised, and continued until contagious stage is passed.
  (*c*) Establishment of Free Dispensaries—open at night—where patients can seek advice, help, and treatment.

(IV) *Appointment of any General Hospital receiving aid from the Consolidated Revenue, or any ward thereof, a place in which persons of both sexes can be placed for treatment.*
  (*a*) Segregation of "dangerous" cases and persons specially liable to communicate the disease.
  (*b*) Legal power to remove to and detain in such hospital such class of patients.

(V) *Transmission of Venereal Disease to be an Offence punishable by heavy fine or imprisonment.*
  (*a*) Such legal provision not to take effect until twelve months from the time the system of gratuitous treatment has been in operation.

II. Educational.

(I) *Formation of a Society for the prevention of Venereal Disease throughout the Commonwealth.*
  (*a*) Branches to be formed in each State, and to include men and women of every profession and social standing.
  (*b*) The object of such Society to be a campaign of *publicity and education.*
  (*c*) Proper distribution of circulars, pamphlets, and other literature, and organisation of lectures, addresses, &c., to the public, parents, young men and women, children, teachers, &c.

  This teaching to be not only moral, but medical in its widest sense, and especially to inculcate the principle of *individual responsibility.*

(II) *State Health Departments* to make a definite and determined effort to awaken and interest the general public in this fight against venereal disease.

(III) *A Course of Venereology to be compulsory in Medical Schools.*

(IV) *Responsibility of the Press, the Pulpit, Physicians, and Parents.*

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PROGRESS REPORT ON THE INVESTIGATION IN SYPHILIS MADE IN THE METROPOLIS OF MELBOURNE FROM 1st JUNE, 1910, TO 31st MAY, 1911.

By Konrad Hiller, M.D., C.B. (Melb.)

I REGRET very much that owing to the amount of work involved, I am unable to give a full report on the Investigation of Syphilis, which was made in the metropolitan area of the city of Melbourne from 1st June, 1910, to 31st May, 1911, and even the present progress report is by no means as searching as I intend at a later date that it shall be. I feel confident, however, that the material I have to place before you to-day will sufficiently interest you to justify me in presenting this paper for your consideration.

I wish, before dealing with the report itself, to describe to you the methods adopted in this investigation, and what provisions were made to avoid errors which might seriously affect its statistical value.

At the beginning of the year 1910 the Government of Victoria decided to set apart a sum of money for the purpose of eliciting how much syphilis was present in Melbourne. It was realised that before any Government measures could be introduced it was necessary to have more certain data of the prevalence of the disease. The chairman of the Board of Public Health (Dr. Burnett Ham) was asked to formulate a scheme which would be feasible and would hold out reasonable hope of success.

Dr. Ham called together an advisory committee, consisting of Professor Allen, Drs. J. W. Barrett, Jeffreys Wood, R. R. Stawell, E. Robertson, and myself, and laid before them the following scheme, which was approved of by the members of the committee present.

To clear the way, syphilis was to be declared a notifiable disease in the Melbourne metropolitan area. The notification was to be impersonal; a circular was to be issued to all the medical men in practice in Melbourne, advising them of what had been done, and asking their co-operation in the scheme which had been formulated. At the same time a copy of what it was intended that men should report as syphilis was to be enclosed. These various manifestations of the disease were arranged as follows:—

(1) Primary.
(2) Secondary.
(3) Tertiary—including gumma, ulcers, fibrosis, eruptions, bone diseases, eye diseases, &c., of certain or doubtful syphilitic origin.
(4) Thoracic aneurism, aortic retroversion.
(5) Apoplexy in young adults.
(6) Locomotor ataxy, general paralysis of the insane, cerebral syphilis.
(7) Congenital and infantile syphilis.
(8) All cases in which a mother—
    (a) gives birth to a syphilitic child;
    (b) has suffered from two abortions;
    (c) has lost three children from disease under 5 years of age.
(9) Any other suspicious case.

This scheme was carried into effect.

By the 1st of June, 1910, every medical man in Melbourne whose named appeared on the register was supplied with a small box containing a few large opsonic tubes, a small phial, together with directions of how to obtain the required amount of blood.

Notification forms were also sent, and requested the following data:—

Sex.
Age.
Nature of lesion.
Any positive evidence.
Duration of anti-syphilitic treatment.
Has blood been taken in this way before?
If so, give approximate date.
Signature and address of sender.
Date.

It was requested that each specimen of blood bearing a distinguishing mark be sent to the Bacteriological Laboratory of the University, and to avoid unnecessary work that the report itself, with the corresponding distinguishing mark, be posted direct to me at the laboratory, instead of at first passing through the hands of the Public Health Department.

I, on my part, undertook to make a Wassermann test on every specimen received, to supply opsonic tubes and phials as required, and on the completion of the test to report the result to the medical man concerned.

From the outset the response to the appeal was large, and increased as time went on. To such an extent did this increase take place that I was forced, in December, 1910, to apply for assistance, and Dr. H. Douglas Stephens kindly consented to co-operate with me. Still further assistance became necessary in February, 1911, when Dr. J. W. Barrett asked that he be allowed to send specimens of blood of all the cases which attended his clinic at the Eye and Ear Hospital.

Side by side with this investigation the test was applied to specimens of blood from post-mortems made at the Children's Hospital, and of which Dr. Lamble received the pathological specimens. Dr. Lamble and I worked independently of one another, and compared results after each had completed his work. The outcome of this will be laid before you by Dr. Lamble.

By the courtesy of Dr. R. J. Bull, Director of the Bacteriological Laboratory, I was enabled to carry out the whole work in his laboratory. During the previous three years I had already been engaged on the Wassermann work, though not on the same large scale, and it was therefore very gratifying to me to continue my investigation at his laboratory without having to go elsewhere.

For some months previous to the beginning of the investigation preparations were made to cope with the work. Rabbits had to be immunised, a stock of guinea-pigs collected, opsonic tubes prepared, and so on.

As regards the testing itself, I feel that it is incumbent upon me to rapidly describe the method by which this was done, since different methods have been employed, with varying results.

I wish, first of all, to state that it was recognised from the outset that the performance of the test itself had to remain in the hands of one man only. At no time was any material which would tend to affect the test allowed out of my own hands. Every test was made by myself. The assistance which I obtained was directed solely towards procuring for me sheep's blood and guinea-pig's blood, pipetting off serum from specimens sent preparatory to inactivation and clerical work.

In the second place, every test was made according to Wassermann's original method or the slight modifications of that method which Wassermann himself employs at the present time. I refer to such things as the use of the alcoholic in place of the watery extract, using one-half cubic centimetre of each diluted ingredient instead of 1 cubic centimetre, &c.

The extract then was obtained from the liver of a known syphilitic fetus. The liver was cut into thin slices and dried. An alcoholic extract was made from this at frequent intervals, and there was thus a practically fresh extract always on hand. The dried liver which was used for the investigation had previously been thoroughly tested side by side with extracts from the liver of other syphilitic fœtuses, and was found to be the most effective.

The patient's serum was always inactivated at 56 deg. Cent. for half an hour, the dilution being 1 in 6.

Complement—guinea-pig's serum—was obtained fresh on every morning that a series of tests were made. At no time was such a serum used that was older than six hours. Even when such an interval elapsed the serum was kept in the ice-chest. The strength used was 1 in 10.

The hemolytic serum was obtained from a rabbit before the investigation began. The method employed was the usual one of injecting increasing quantities of well-washed red corpuscles of the sheep until the required strength had been obtained—in this instance 1 in 5,000. The rabbit was then exsanguinated, the blood being caught up in a large Petri's dish. After the serum had separated from the clot it was drawn up into small tapered glass tubes, inactivated and stored in the ice-chest. The whole of this process was, of course, carried out under aseptic conditions. The strength of this serum had not in any way diminished at the end of fifteen months, and is in use at the present time. It was used in the strength of 1 in 2,400 to 1 in 2,600, according to the preliminary test. This preliminary test was invariably made prior to the commencement of the real series of tests.

The sheep's corpuscles were obtained every morning that a series of tests were made from sheep kept at the laboratory for the purpose. The emulsion used was 5 per cent. in saline.

The saline was made by myself, and was ·9 per cent. in distilled water.

As already stated, the quantities of the diluted ingredients were always a half c.c.

Results were read off one hour to one and a half hours after the beginning of the second incubation period. The final reading was made on the following morning.

As regards these readings, positive results were only given as such where there was practically no hemolysis. Tubes which, although presenting a definite deposit of corpuscles, still showed decided hemolysis were designated partial reactions. Although I have classed these separately in the statistical table which follows, no importance has been attached to them.

My own views of these reactions are that when taken in conjunction with the clinical appearances they may be of the greatest value. I have been able to watch the effect of anti-syphilitic treatment on some such cases, and have frequently seen the manifestations of symptoms subside. I need merely mention here two cases which were of striking interest from this point of view. The first was the case of a male showing marked small ulcers of the tongue, which would not respond to ordinary treatment. The Wassermann reaction was partial. He denied infection, but admitted having run risks. On internal treatment with iodide of potassium and mercury the condition rapidly resolved. Treatment was continued for five months, and then stopped. Six months afterwards he presented himself with a typical nodular syphilide of the chest wall, which again yielded to anti-syphilitic treatment.

The second was also a male, who was assisted into the room by his sons. He complained of great vertigo, weakness, and some soreness of the back of the neck. Without entering into the examination, it may be said that he showed marked cerebellar symptoms, and the serum reaction was partial. Under anti-syphilitic treatment symptoms disappeared, and recently he was seen walking alone in the street. I might say that the condition had been a steadily progressive one for the previous five months.

One more case to illustrate this point. The patient was a male, suffering from severe nocturnal headaches and some mental symptoms. He denied venereal infection, and showed very few neurological signs. The serum reaction was partial. Some improvement took place under treatment; but he then got a sudden attack, which was diagnosed as a basal affection, probably because of its progressive character a thrombosis of the basilar artery. He succumbed thirty-six hours afterwards, confessing to a near relative before his death that he was being treated for syphilis when first seen. A post mortem was allowed, and the diagnosis verified. Sections of the basilar artery showed typical endarteritis obliterans.

I have given these cases as illustrating the value of a partial reaction. Other investigators, however, hold the view that partial results are not to be relied upon. It has, therefore, been insisted upon in this work that such results, from a statistical point of view, must be ignored.

As regards the response of medical men to the appeal for co-operation, as far as I can ascertain there are 350 medical men and women practising in Melbourne. Of these 110 reported cases. This number includes those who sent only two or three specimens, and when analysing the report this fact has, of course, to be borne in mind. I believe the chief reasons for such a comparatively small response were the difficulty which is experienced in obtaining sufficient blood—this is especially the case in children—and the time it requires to collect a specimen. A large number of specimens were received which had to be cast aside because of insufficient quantity. No account of these has, of course, been taken in this report.

Total number of cases examined and recorded to date, 3,190.

Results according to the Wassermann test :—

Positive.	Partial.	Negative.
1,320	300	1,570

Results tabulated according to whether the cases were private or from a public institution :—

	Positives.	Partial.	Negative.	Total.
Private cases	508	121	765	1,394
Public institutions ...	812	179	805	1,796

Detailed results :—

(1) *Primary Stage.*

	Positives.	Partial.		Negative.		Total.
		Untreated.	Treated.	Untreated	Treated.	
Private cases	27	3	...	19	2	51
Public institutions ...	35	2	1	17	5	60
Total number examined ...	...	...	...	...	...	111

Results—Positive, 62; Partial, 6; Negative, 43.

	Positives.	Partial.		Negative.		Total.
		Untreated.	Treated.	Untreated.	Treated.	
(2) *Secondary Stage.*						
Private cases	93	...	2	5	9	109
Public institutions ...	105	2	3	10	15	135
Total number examined	...	...	...	...	...	244
Results—Positive, 198; Partial, 7; Negative, 39.						
(3) *Tertiary Stage.*						
Private cases	193	14	28	48	116	399
Public institutions ...	187	4	20	44	52	307
Total number examined	...	...	...	...	...	706
Results—Positive, 380; Partial, 66; Negative, 260.						
(4) *Diseases of the Aorta and Aortic Valves.*						
Private cases	17	1	2	11	6	37
Public institutions ...	34	2	...	16	...	52
Total number examined	...	...	...	...	...	89
Results—Positive, 51; Partial, 5; Negative, 33.						
(5) *Hemiplegia in young adults.*						
Total number examined	...	...	...	...	...	2
Results—Both negative; both cases from public institutions. One treated, the other not.						
(6) *Parasyphilis.*						
Private cases	31	1	3	5	14	54
Public institutions ...	55	4	2	19	7	87
Total number examined	...	...	...	...	...	141
Results—Positive, 86; Partial, 10; Negative, 45.						
(7) *Congenital Syphilis.*						
Private cases	27	1	1	24	1	54
Public institutions ...	116	44	4	166	12	342
Total number examined	...	...	...	...	...	396
Results—Positive, 143; Partial, 50; Negative, 203.						
(8) *Mothers who have borne syphilitic children—had two or more miscarriages, &c.*						
Private cases	17	7	4	22	5	55
Public institutions ...	30	6	...	27	...	63
Total number examined	...	...	...	...	...	118
Results—Positive, 47; Partial, 17; Negative, 54.						
(9) *Fathers of syphilitic children.*						
Total number examined	...	...	...	...	...	4
Results—Positive, 3; Negative, 1. Three private and one institution case.						
(10) *Any other suspicious cases.*						
Private cases	100	45	9	449	28	631
Public institutions ...	250	75	10	409	4	748
Total number examined	...	...	...	...	...	1,379
Results—Positive, 350; Partial, 139; Negative 890.						

PERCENTAGE Results Compared with other Investigators.

Primary.

Total.	Positive.	Partial.	Negative.	Percentage.
111	62	6 (1 treated)	43 (7 treated)	56

Other investigators vary between 50 and 71·6 per cent. for 50 cases or more.

Secondary.

244	198	7 (5 treated)	39 (24 treated)	81

Other investigators vary between 74 and 100 per cent. for 100 cases or more.

Tertiary.

706	380	66 (48 treated)	260 (167 treated)	54

Other investigators vary between 63 and 100 per cent. for 25 cases and more.

Thoracic aneurism and Aortic retroversion.

89	51	5 (2 treated)	33 (6 treated)	57·4

	per cent.
33 cases of thoracic aneurism, of which 30 were males and 3 females. Positive, 22; Partial, 2 (both treated); Negative, 9 (5 treated)	66·6
56 cases of aortic retroversion, of which 49 were males and 7 females. Positive, 29; Partial, 3 (1 treated); Negative, 24 (none treated)	52

Hemiplegia in young adults.

Total.	Positive.	Partial.	Negative.	Percentage.
...	...	...	...	...

Parasyphilis.

141	86	10 (5 treated)	45 (21 treated)	61

	per cent.
59 cases of tabes, of which 54 were males and 5 females. Positive, 34; Partial, 2 (1 treated); Negative, 23 (12 treated)...	57·6
34 cases of cerebral syphilis, of which 33 were males and 1 female. Positive, 14; Partial, 7 (3 treated); Negative, 13 (5 treated) ...	41
48 cases of G.P.I., of which 38 were males and 5 females (5?). Positive, 38; Partial, 1 (treated); Negative, 9 (4 treated) ...	79

(Of the 9 negative cases 6 were ? G.P.I.)

Congenital Syphilis.

Total.	Positive.	Partial.	Negative.	Percentage.
396	143	50	203	36

Mothers who have borne syphilitic children—had two or more miscarriages, &c.

118	47	17	54	40

Any other suspicious cases.

1,379	350	130	890	39

As already mentioned, this report is not complete. About 5,550 cases were examined; 550 of these will be excluded, as they were taken from Dr. J. W. Barrett's clinic at the Eye and Ear Hospital, irrespective of whether they presented syphilitic or suspected syphilitic lesions or not; 1,900, approximately, out of the 5,000 cases which remain gave positive results. For the final report Dr. H. Douglas Stephens has promised to give the clinical aspect of those that were sent from the Children's Hospital, and those from the Melbourne Hospital will be dealt with by the registrars of that institution.

It would be premature for me at the present time to draw any conclusions from the report just submitted. This, however, I am able to say—that the percentages of positive results which I obtained in the various stages of the disease are low when compared with those of other investigators where comparison is possible.

HISTOLOGICAL STUDIES COMPARED WITH THE WASSERMANN REACTION.

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Many years ago Fournier,[1] basing his conclusions mainly on clinical grounds, said that inherited syphilis was the great underlying cause of infant mortality, and since his day an increasing body of pathologists, basing their conclusions on certain macroscopic and microscopic appearances, have come to hold the same view; many clinicians, however, still doubting both the accuracy of the conclusions and the reliability of the standards from which they have been drawn.

The advent of the Wassermann reaction seemed to offer a means of obtaining further statistical information bearing on this question, and also a reliable method of checking the value of the criteria which have been adopted; accordingly I have examined miscrosopically sections of the liver and spleen in 100 cases coming to autopsy at the Children's Hospital, Melbourne, the series being practically consecutive, only those being discarded in which putrefactive changes introduced a possible source of error in histological or serological findings.

The blood in each of these cases was submitted to independent examination by means of the Wassermann reaction, and in all but a few cases it was obtained post mortem either from the heart or the subclavian vein, the work of Nauwerck and Weichert[2] having shown results obtained under similar conditions to be reliable; while Obregia and Bruckner,[3] who examined cerebro-spinal fluid kept in unsterilised vessels teeming with putrefactive organisms at intervals during a period of over six months, found that the syphilitic antibody is extremely resistant to putrefaction, a positive reaction being weakened or transformed into a negative one in but a few cases.

In no case was the result of the Wassermann reaction known until the histological sections had been examined and the findings noted, a diagnosis of syphilis being made if both liver and spleen showed the presence of interstitial fibrosis of varying age and activity, together with a thickening of the coats of the smaller arteries and activity of their endothelium. No case was regarded as positive which did not show the presence of these vascular changes. Schultz[4], who recently made a series of comparative studies of tissues stained by silver methods to show the presence of S. pallida and ordinary histological preparations, regarded these vascular phenomena as the essential indication.

A survey of the comparative results in this series after they were brought together has convinced me that the spleen, which is so rapidly and markedly affected by most conditions of general toxæmia, is not a thoroughly reliable guide in the histological diagnosis of syphilis. Its smaller vessels, particularly the central arteries of the malphigian bodies, readily shows signs of irritation in association with local suppuration elswhere in the body, in the absence either of a positive Wassermann or of appearances suggestive of syphilis in other organs, while some degree of overgrowth of the fibrous frame-work was present in all but two cases in the series. On the other hand, however, the presence of the histological appearances adopted as diagnostic of syphilis in the liver may be regarded as very reliable, as the following figures will show.

Of the 100 cases examined, 54 gave a positive Wassermann reaction, 17 a partial reaction, while 29 were negative. The histological findings agreed very closely with these, for the whole 54 cases with a positive serum action were returned as positive histologically, while of those giving a partial reaction 14 were diagnosed as certainly positive, and 3 as doubtful, but probably positive; but further details of the histological peculiarities of these cases will presently be mentioned. Turning now to the cases giving a negative serum reaction—29 in all—of these, 14 cases were diagnosed as positive histologically, 5 as doubtful, and in 10 cases the histological and serum diagnosis agreed. Now, of the 14 cases diagnosed as positive, 5 had definite clinical manifestations of syphilis, and 3 of these were under mercurial treatment at the time of their death, thus leaving 9 cases with an absolute disagreement between the histological and serum diagnosis and without clinical signs in support of the histological diagnosis. But Rubens[5] has shown that many cases with inherited syphilis of a latent type only present a positive Wassermann reaction coincidentally with the onset of clinical signs, previous results having been negative, and it is more than probable that these 9 include some such cases.

Appended are the results in tabular form:—

Wassermann Reaction.		Histological Diagnosis.			
		Positive.	Doubtful.	Negative.	Total.
Positive	54	54	...	...	54
Partial	17	14	3*	...	17
Negative	29	14†	5	10	29
Total	100	82	8	10	100

* Probably positive. † Five with definite clinical manifestations.

Now, a consideration of any of the recorded investigations shows that many cases of syphilis do not give a positive Wassermann reaction, and some correction must be applied to any statistics based on this reaction. The Committee in charge of the Victorian investigation into the prevalence of syphilis have adopted as the basis for their report a correction of 40 per cent (*i.e.*, that 60 positives represent 100 cases of syphilis). If this be correct, it is obvious that there is a vastly greater measure of agreement between the histological standards of syphilis adopted and the Wassermann reaction than there is between clinical standards and the reaction.

But it may be objected that these results taken from cases chiefly of acquired syphilis do not apply to the inherited form of the disease, and, although the figures are on a small scale, Kaliski[7] concludes that 98 per cent. of infants and 50 per cent. of children with hereditary syphilis give a positive reaction. The necessary correction for this class of case then lies somewhere between 2 per cent. for infants and 50 per cent. for older children; so that, even adopting these figures, the results of the present investigation are well within the limits suggested by Kaliski.

The Partial Cases.

A great difficulty is always experienced in the interpretation of partial reactions, but a consideration of the histology of the cases giving such a reaction in this series seems to make it clear that a partial reaction means the presence of a syphilitic process in a quiescent or retrogressing stage, for the majority of these cases show a retrocedent tendency, as evidenced by—

(a) hyaline degeneration of the thickened wall of the small arteries; or

(b) the absence of signs of activity in the over-grown fibrous tissue of Glisson's capsules in the liver.

Again, three of these cases which had given a definite history of syphilis, and had been under treatment for short periods, all showed very definitely this retrocedent tendency.

Syphilis as the great Underlying Cause of Infant Mortality.

Coming now to look more in detail at the effect of syphilis in causing infant mortality. If we put out of consideration for the time being all those cases in which the histological findings did not absolutely agree with the serum reaction or clinical phenomena, the facts are still very striking, for to the 54 cases giving a positive reaction must be added 5 cases with definite clinical evidence of syphilis but giving a negative Wassermann reaction, and 3 similar cases giving a partial reaction, thus bringing the total number of undoubted cases of infection up to 62 (per cent.). Fournier[8] says that "many of these children carry death in their birth," and certainly the process must start in some cases very early in intra-uterine life. For example, case No. 20 in this series shows in the liver of a child fourteen days after birth large tracts of old fibrous tissue completely inactive and undergoing retrogressive changes. So it is that very many of these cases die early during the first year. Of 44 cases in this series dying during the first twelve months, all, without exception, were diagnosed histologically as syphilitic—that is to say, all were "fibrotic"; and among these 44 infants, 26 gave a positive Wassermann reaction and 10 a partial reaction, while 8 (of which 3 were clinically definite cases of syphilis) gave a negative reaction.

Cases Dying under Twelve Months.

Total number	44
Histologically positive	44
Wassermann positive..	26
Wassermann partial	10
Wassermann negative	8*

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* 3 definite clinical cases of syphilis.

But further light is thrown on the problem of the influence of syphilis in bringing about early death by a consideration of the ages and of the immediate cause of death in the 10 cases in which there was neither clinical, serological, nor histological evidence of syphilis present. Only 3 of these cases were under 4 years of age, and 4 of them were over 8, while in every case death was due to severe illness such as sarcoma of kidney (1 case), acute nephritis (1 case), cerebral abscess (2 cases), general peritonitis after operation for gastrostomy (1 case), general tubercle (1 case), rheumatic endocarditis (2 cases), broncho-pneumonia, with death on the fourteenth day (1 case).

These histories stand in marked contrast to those of the previous series (*i.e.*, cases dying under one year), in the majority of which death was ascribed to gastro-enteritis, broncho-pneumonia of short duration, rapid generalised tubercle, or mild infective disorders, while a few followed small surgical operations.

The Resistance of the Congenital Syphilitic.

Not only is syphilis responsible for much infantile mortality, but also it leads to diminished resistance to mild infective diseases, and to many sudden deaths with only slight lesions to be found post mortem. The present series abounds with illustrative examples in which the organs showed the characteristic changes and the Wassermann reaction was positive. For instance, one may quote case No. 20* a child 14 days old, with sudden collapse and death three hours after the complete cessation of a small umbilical hæmorhage, the only lesion found post mortem being a small hæmorrhage into the thymus gland; or, again, cases 39, 73, 76, with death in the first twenty-four hours from the onset of a broncho-pneumonia, with only slight lesions post mortem; or, as an example of yet another class, case No. 92, ill seven days with cough, epistaxis, and signs of cardiac dilatation, the only lesion found post mortem being a dilated heart with no valvular lesion; or, again, case No. 35†, with death after a short period of coma and acetonuria, the only gross lesion noted post mortem being œdema of the brain.

The Relation of Generalised Tubercle and Gastro-Enteritis to Fibrosis and Vascular Changes in the Liver.

It is commonly objected by those who do not accept the views advanced in this paper that the histological standards adopted are merely those of chronic inflammation, and might be caused by any chronic infective process such as tubercle or gastro-enteritis. That such may be the case in the spleen I have already stated; but that general vascular changes or fibrosis are produced by tubercle is contrary to all experience, not only in children, but in cases dying in adult life. The material in this series bearing on the point is too limited to warrant much attention, but all the cases of generalised tubercle, with one exception, gave a positive or partial Wassermann reaction, and showed generalised vascular changes; or, in other words, they were cases of tubercle grafted on to congenital syphilis, and running a downward course because of the devitalising influence of the inherited disease. The negative case (No. 17) did not show generalised arterial change in the liver, the only arteries affected being in the neighbourhood of the tubercular foci.

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* The numbers refer to appended table. † Wass. R. negative—clinical history of syphilis.

The relationship, however, between gastro-enteritis and syphilis and between gastro-enteritis and fibrosis and fatty changes in the liver is by no means so clear.

An examination of the appended table will reveal many cases showing chronic ulceration of the mucous membrane of the bowel with fibrosis of the outer coats and arterial change, in association with a positive Wassermann reaction and the following changes in the liver:—

1. Fibrosis in Glisson's capsules and between the cells.
2. Arterial changes of the "syphilitic" type.
3. Fatty degeneration.
4. Lysis of liver cells.

The last two changes were often so extreme as to leave few normal liver cells visible in any microscopic field. The difficult question to settle is whether the toxins elaborated in the diseased bowel are the direct cause of these latter degenerative changes in the liver, or whether the changes in the liver and bowel are but part of the same syphilitic process, the gastro-enteritis being merely an expression of metabolic disturbances consequent on abolished or diminished function of the liver.

The material available is not enough to settle the point, which needs further post mortem, clinical, and experimental investigation. It does, however, provide sufficient evidence to justify one in regarding the latter view as at least probable, chiefly because, in the majority of cases, the fatty change in the liver has been apparently of slow evolution and because the vascular changes affect the small arteries to a very much greater extent than the branches of the portal veins.

The Relation of Microscopic Appearances to Clinical and Post-Mortem Standards.

Although it has been pointed out that in many cases the process must start quite early in intra-uterine life, in others the inception is apparently delayed till a much later period of gestation, or even till some time after birth. Such cases may not present much enlargement of the organs nor gross fibrosis at the post mortem examination, and may easily be missed till microscopic sections are examined, the growth of new tissue not being sufficient to cause an alteration in the consistence of the organ sufficient to be evident to the examining finger. But more striking still is the fact that over half the cases do not present at all the classical picture of congenital syphilis, the only hint given to the clinician in many of them being the poor resistance of the child, and the abnormal course run by some inter-current complaint. Schultz[9], who also draws attention to this fact, was struck by the frequency with which he found S. pallida in sections of the organs of chidren dying soon after birth with no clinical manifestations of the disease, and points to the frequency with which the diagnosis is missed by both the clinician and the pathologist, and says that "if one requires complex changes in lungs, liver, and spleen he will miss many luetic cases in young children."

A general consideration of the material dealt with leaves nothing to be added to the description of the morbid anatomy of congenital syphilis published by Professor Allen[10] in 1904, but it establishes the accuracy of the criteria he then enunciated, and consequently of the conclusions he then drew from post mortem experience with regard to the prevalence of syphilis. He then stated: "My figures must be taken for what they are, and no more general meaning must be read into them. They do not apply to the living hospital patients; they do not apply to the general community." The same remarks apply, but with greater force, to my own figures; first because the Children's Hospital, perhaps, less than any other may be taken as an index of the general population; and more important still, as Fournier, Schultz, and others have pointed out, death rapidly thins the ranks of the congenital syphilitic in the first few years of life, and so the figures derived mainly from children under 5 years must necessarily show an abnormally large percentage affected with the disease. Nevertheless, whether they belong to a hospital population or not, the loss of so many children is extremely serious from the national standpoint. The fact that out of the 100 cases now submitted considerably more than half were syphilitic should merit the most careful consideration by those interested in public health.

In conclusion, my best thanks are due to Dr. Hiller (who performed the Wassermann reactions) for many suggestions, but especially to Dr. H. D. Stephens, Pathologist to the Children's Hospital, Melbourne, who obtained for me both the opportunity of carrying out the investigation and also access to the clinical records of the cases examined.

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No.	Age.	Histological Appearances.			Serum Reaction.	Mercurial Treatment.	Clinical History.
		Liver.	Spleen.	Bowel.			
1	10 days ...	Capsule not greatly thickened. Increase in fibrous tissue in Glisson's capsule. Some pericellular fibroblastic infiltration. Vessels thick-walled, endothelium active. Definite periarteritis.	Capsule thickened. Vessels thick walled with active endothelium. Perivascular accumulations of fibroblasts.	Not examined	+ (ante-mortem.)	....	Premature child. Mother dying of phthisis. Regarded clinically as typically syphilitic.
2	9 months	Capsule thickened. Increase in fibrous tissue in Glisson's capsules. No active fibrosis. Vessels thick-walled, endothelium active.	Great increase in fibrous stroma. Vessels thick-walled. Endothelium active. Some fibroblasts in pulp.	Not examined	+	10 days ...	Diarrhœa and vomiting intermittent for 6 months. Marked syphilitic facies. Family history—6 miscarriages, 1 still-born child, 3 others apparently healthy.
3	2 years ...	Thickened capsule. Some fatty degeneration. Great increase in fibrous tissue in Glisson's capsule, chiefly old; vessels thick-walled with active endothelium, though vascular change is patchy.	Diffuse fibrosis, chiefly old. The vessels are thick-walled, and show hyaline change. The endothelium is active.	Not examined	—	....	Mongol idiot. Ricketty, with open frontanelle. Death from broncho-pneumonia. 10 days' illness.
4	1 month ...	Increase of fibrous tissue and fibroblasts in Glisson's capsules. Some pericellular fibroblasts. Vessels are thick-walled. Endothelium active. Some congestion.	Some increase in fibrous stroma. The vessels are thick-walled, showing both endarteritis and periarteritis.	Not examined	Partial ...	....	Hypertrophic stenosis of pylorus.
5	3 months	Capsule slightly thickened. Extreme fatty degeneration. Marked fibroblastic activity in Glisson's capsules. Vessels thick-walled, with very active endothelium. Perivascular cell accumulation.	Capsule thickened. Vessels thick-walled with marked endarteritis and periarteritis. Diffuse fibrosis.	Not examined	+	Treatment for some months.	Marked syphilitic stigmata. Gastro-enteritis.
6	3 years ...	Well marked multilobular fibrosis. Some activity at present. Vascular changes irregular. Doubtful, but probably positive.	Vessels thick-walled. Endothelium active. Very little perivascular cell accumulation. Capsule thickened and some increase in fibrous stroma.	Not examined	—	?	Nephritis.
7	17 days ...	Cirrhosis active and chiefly pericellular. Vascular changes patchy, but some vessels are thick-walled, with active endothelium and perivascular irritation.	Diffuse fibrosis and fibroblastic infiltration. Vessels thick-walled, with active endothelium and definite periarteritis.	Not examined	+	....	Constipation—bowels never opened naturally since birth, never thriven, small child, rash on buttocks and labia.
8	....	Active fibroblastic infiltration in Glisson's capsules and between cells. Vessels show very active endothelium and fibroblasts in their middle and outer coats.	Capsule slightly thickened. Diffuse fibroblastic infiltration. Vessels thick-walled, fibroblastic infiltration of outer coat. Great activity of endothelium.	Not examined	—	....	Not obtainable.

No.	Age.	Histological Appearances.			Serum Reaction.	Mercurial Treatment.	Clinical History.
		Liver.	Spleen.	Bowel.			
9	12 years ...	Active fibro-blastic infiltration of Glisson's capsules. Vascular changes irregular. Endothelial activity more marked than changes in outer coats.	Capsule thickened. Large areas of caseation. Increase in fibrous framework. Vessels—endothelium active, fibrosis of outer coats, perivascular cell accumulation.	Not examined	+ (ascitic fluid.)	....	Hodgskin's disease.
10	1 year and 2 months.	Some increase in fibrous tissue in Glisson's capsules. Active fibroblasts. Microscopic tubercles. Vascular changes are definite.	Capsule thickened, irregular overgrowth of fibrous tissue of stroma. Vascular changes irregular, some showing thick walls and active endothelium	Not examined	+	....	Generalised tubercle.
11	10 years ...	Very little increase of fibrous tissue. No vascular changes.	Capsule thickened. Vessels are thick-walled and hyaline. No active fibrosis or peri-articular irritation. Some activity of endothelium of arteries	Not examined	—	....	Cerebral abscess sequent. On middle ear suppuration.
12	4 years and 9 months	A little fibroblastic activity in Glisson's capsules. No changes in smaller vessels. Some fibroblastic infiltration and endothelial activity in the larger arteries. No engorgement.	Capsule thickened. Some increase in fibrous stroma. Patchy thickening of vessels with endarteritis.	Not examined	—	....	Pericarditis } 1 year's Endocarditis } duration. Pneumonia.
13	....	Liver—multilobular and pericellular fibrosis. Vessels fibrosis of outer coats. Endarteritis well marked.	Thickened capsule. Increase of fibrous framework. Vessels thick-walled, with well-marked endarteritis and perivascular fibrosis.	Not examined	+	....	Diabetes. Rapid onset of coma and death. No suspicion of syphilis clinically.
14	....	Capsule greatly thickened. Increase of fibrous tissue of varying age in Glisson's capsules. Pericellular cirrhosis. Vessels thick-walled. Endarteritis, but very little periarteritis.	Well-marked fibrosis and thickening of walls of arteries which have undergone hyaline changes. Patchy endarteritis.	Not examined	+		No history obtainable, but gland sent for examination with specimens, was an old organising gumma.
15	....	Thick capsule. Great increase of fibrous tissue in Glisson's capsules. Foci of active growth. Vessels thickened, some showing hyaline change. Endarteritis.	Capsule thickened. Great increase in fibrous stroma. Vessels hayline degeneration of thickened walls. Endothelium active.	Not examined	Partial ...	Treated in Out-patients' Dept.	Regarded as a syphilitic; anæmic.
16	9 years ..	Slight thickening of capsule. Slight fibrosis. No vascular changes.	Some fibrosis. Thickening of coats of all the small vessels. Hyaline change in outer coats. A few vessels show activity of endothelium.	Not examined	—	....	Purulent meningitis. Duration of illness, 3 days. Considered at post-mortem to be probably syphilitic. Thymus enlarged. Liver and spleen enlarged and firm.

No.	Age.	Histological Appearances.			Serum Reaction.	Mercurial Treatment.	Clinical History.
		Liver.	Spleen.	Bowel.			
17	1 year and 4 months.	Capsule thickened, slight increase of fibrous tissue in Glisson's capsules. Some microscopic tubercles. No general vascular changes. Slight fatty degeneration.	Capsule greatly thickened. Areas of fibrosis with more active cell infiltration. Vessels—walls thickened and hyaline. Marked endareritis.	Not examined	—	....	Tubercular lymph glands. History incomplete.
18	3 weeks ...	Active fibroblastic infiltration of Glisson's capsules and between the cell columns. Vessels thick-walled, with very active endothelium.	Capsule thickened. Increase in fibrous tissue of framework. Active fibrosis. Vessels show thick walls with definite endarteritis.	Not examined	+	....	Father had syphilis. Child died of cellulitis of leg in 36 hours.
19	5 months	Thickening of capsule. Multilobular and pericellular cirrhosis. Vessel walls thickened. Endothelium active. Some fatty degeneration.	Great increase in fibrous stroma. Vessels thickened. Perivascular fibrosis. Endarteritis with a tendency to thrombosis in some of smaller vessels.	Not examined.	+	....	Cerebro-spinal meningitis. Ill 3 weeks. Terminal broncho-pneumonia.
20	2 weeks ...	Increase in fibrous tissue in Glisson's capsules ,but no sign of activity. Vessels thick-walled, with hyaline change in walls. Some proliferation of endothelium. Active pericellular fibroblastic infiltration.	Diffuse fibrosis more active than in the liver. Vessels thick-walled, showing little degeneration and active endothelium.	Not examined	+	....	Umbilical hæmorrhage with sudden collapse 3 hours after hæmorrhage had been completely controlled. Petechial hæmorrhages in pleura, hæmorrhage into thymus.
21	....	Increase in fibrous tissue in Glissons' capsules. Vessels thick-walled, with marked endarteritis. Pericellular fibroblastic infiltration. Slight fatty degeneration.	Not examined	Not examined	+	....	
22	....	Not examined	Capsule slightly thickened. Marked fibrosis and thickening of vessels, which show definite endarteritis and periarticular fibrosis.	Not examined	+	....	Sub-acute abscess of left kidney.
23	....	Capsule not thickened. Well marked, multilobular and pericellular fibrosis; great thickening of vascular walls, and active round cell infiltration around vessels—marked endarteritis.	Capsule greatly thickened. Diffuse fibrosis. Thickening and hyaline change in vascular walls. Endothelium active.	Not examined	Partial ...	....	
24	4 years and 6 months.	Fatty liver with active fibrosis of and thick vessels, but no endothelial activity. No periarticular cell accumulation.	Some fibrosis with thick-walled vessels, but no endothelial activity.	Not examined	—	....	Not syphilitic. Old æsophageal stricture following KOH. Gastrostomy and general peritonitis. History of gastro-enteritis intermittent for some years.

No.	Age.	Histological Appearances.			Serum Reaction.	Mercurial Treatment.	Clinical History.
		Liver.	Spleen.	Bowel.			
25	10 days ...	Fibroblastic infiltration between cells. Endothelium of capillaries very active. No great thickening of arteries.	Capsule thickened. Great fibrosis, vascular thickening and endarteritis.	Atrophic	Partial ...	....	Miserable child—red about anus. Fed on condensed milk. Hæmorrhagic effusion on dura under parietal bone. Umbilicus not healed.
26	....	Fibrosis. Thickening of vascular walls with marked periarteritis and endarteritis.	Diffuse fibrosis. Marked thickening of walls of all small vessels. Endothelium active. Periarterial fibrosis.	Not examined	Partial ...	....	
27	3 years and 4 months	Slight increase in fibrous tissue in Glisson's capsules of varying age. Vessels show thickening of outer coats and activity of endothelium.	Capsule thickened. Diffuse fibrosis. Vessels as in ilver.	Not examined	—	....	
28	9 months	Increase in fibrous tissue in Glisson's capsules. Active foci of fibroblasts among older fibrous tissue. Vessels thick-walled. Endothelium active.	Not examined	... Not examined	Partial ...	....	Tubercular meningitis, generalised miliary tubercle. Nothing suggestive in family history.
29	2 months	Fatty liver, with some multilobular and pericellular cirrhosis. Activity round vessels, which are thick-walled. Endothelium active.	Capsule not thickened. Diffuse fibrosis. Vascular walls thickened. Endothelium active. Patchy periarteritis.	Not examined	+	....	Congenital syphilitic asthenia. Gastro-enteritis since birth.
30	4 months	Some fibrosis, chiefly pericellular. Vessels thick-walled with marked endarteritis.	Great thickening of the capsule. Diffuse fibrosis, with marked thickening of vessel walls and endarteritis.	Marked ulceration of colon. Sub-acute inflammation with old syphilis of small bowel.	+	....	Catarrhl enteritis and colitis for 1 month; cough 7 days before death. Definite signs of broncho-pneumonia 2 days before.
31	7 years ...	Some old fibrosis, with more active foci. Pericellular fibrosis and fibroblasts. Vessel walls hyaline and thickened. Endothelium active.	Diffuse fibrosis with thick-walled vessels. Endothelium active.	Not examined	+	....	Rheumatic fever when æt. 4. Some endocarditis then. 3 weeks before admission sudden left-sided heimplegia, without loss of consciousness. Some mitral endocarditis. Sudden death. Post-mortem pulmonary œdma, with right-sided cortical hæmorrhage.
32	7 months	Slight increase of fibrous tissue in Glisson's capsule. Thick-walled vessels with endarteritis and periarterial cell accumulation.	Diffuse active fibrosis. Vessels thick-walled and hyaline. Endarteritis.	Not examined	—	?	Rash since birth. Considered syphilitic. Death from broncho-pneumonia. Ill 2 days. Sudden heart failure. History of 1 miscarriage previous to her birth.

No.	Age.	Histological Appearances.			Serum Reaction.	Mercurial Treatment.	Clinical History.
		Liver.	Spleen.	Bowel.			
33	1 year ...	Slight increase of fibrous tissue in Glisson's capsule of varying age. Vessels show endarteritis. Thickening of walls, some hyaline, some showing active infiltration. Slight fatty degeneration.	Thickening and cellular infiltration of capsule. Fibroblastic infiltration of coats of all smaller vessels, with marked endarteritis and periarteritis.	Not examined	—	....	Five others in family. 2 miscarriages, 1 stillborn; coryza for 3 months; mentally deficient; chokes when taking food. Breast fed. Died suddenly. Only lesion noted post-mortem was œdema of brain.
34	9 months	Fibro-fatty, active fibro-blastic infiltration, chiefly in Glisson's capsuels. Vessels thick-walled. Endothelium active. Miliary tubercles throughout section.	Diffuse fibrosis. Vessels thick-walled. Endothelium active. Periarticular cell accumulation.	Not examined	Partial ...	Treatment for some weeks in O.P.	Congenital syphilitic. Generalised tubercle. Both parents gave + Wasserman R. 2 miscarriages; 5 children, all with positive Wassermann reaction. Ill 2 days before death with broncho-pneumonia.
35	11 years	Some increase in fibrous tissue in Glisson's capsules, both old and active. Vessels thickened. Marked endothelial activity. Active pericellular fibro-blastic infiltration.	Some thickening and hyaline change in walls of small arteries. Endothelial activity. Perivascular fibrosis.	Not examined	—	....	Ill 1 month with pericarditis, endocarditis. Death from broncho-pneumonia.
36	11 years ..	Marked multilobular and pericellular cirrhosis. Vessels show great thickening of walls, only a few showing any signs of activity.	Extreme diffuse fibrosis, very little trace of spleen pulp being visible. Vessels for most part have hyaline walls. A few show active endothelium.	Not examined	+	....	Syphilitic ænemia. Enlarged spleen and liver. Death following copious hæmatemesis and malæna. Said to have been well till a few days before death.
37	2 years and 6 months.	Multilobular fibrosis, but condition of vessels is doubtful. A few are thick-walled and hyaline, a few show endothelial activity.	Capsule slightly thickened. No great fibrosis, but vascular changes are well marked.	Not examined	+	....	Death from pneumonia on the third day. Nothing suggestive of syphilis clinically, or in family history.
38	7 years ...	Multilobular cirrhosis Vessels thick-walled. Endothelium active. Some perivascular fibrosis.	Capsule thickened. Slight fibrosis. Vessel walls thickened and endothelium active.	Not examined	+	....	No suspicion of syphilis. Ill 17 days. Measles. Lobar-pneumonia, empyema.
39	11 weeks	Multilobular and pericellular fibrosis, not showing much sign of activity. Slight fatty degeneration. Vessels have thick walls, with hyaline degeneration. Patchy endothelial activity.	Slight fibrosis. Great engorgement and hæmorrhage. Vessels thick-walled and hyaline. Endothelium generally acactive.	Not examined	+	....	A seven-months child. Death from broncho-pneumonia on third day.

No.	Age.	Histological Appearances.			Serum Reaction.	Mercurial Treatment.	Clinical History.
		Liver.	Spleen	Bowel.			
40	13 years ...	Marked multilobular and pericellular fibrosis. Vessels very thick-walled. Endothelial activity patchy in distribution.	Capsule slightly thickened. Diffuse fibrosis. Larger vessels have hyaline walls. Active fibro-blastic infiltration with endarteritis and periarteritis on smaller ones.	Not examined	+	....	Born in Kew Asylum Deaf and dumb. Syphilitic purpura. Syphilitic. ænemia. Gastro-enteritis. Ill 1 month.
41	1 month ...	Fibro-fatty. Extreme degree of fatty change. Vessels thick-walled with active endothelium. Perivascular cell accumulation.	Capsule thickened; increase of fibrous stroma. Vessels as in liver.	Not examined	+	....	Syphilitic family. Others under treatment in Outpatients' Department. Death from chronic suppurative meningitis. 3 weeks' illness.
42	4 months	Multilobular and pericellular fibrosis of liver. Vessels show marked endothelial activity.	Diffuse fibrosis. Vessels thick-walled with active endothelium.	Not examined	—	Hyd. c Cret. and Grey oil for 2 months.	Regarded as syphilitic. Mother looks syphilitic. 3 previous children died in infancy. Gastro-enteritis of 3 months' duration.
43	8 months	Fibro-fatty. Some active foci of fibrosis multilobular and pericellular, with hyaline change in coats. Endarteritis.	Capsule greatly thickened. Diffuse fibrosis. Vessels as in liver.	Engorgement of sub-mucous vessels. Slight ulceration of colon.	—	....	Entero-colitis intermittent Fatal attack only lasted 24 hours. ? Status lymphaticus. Thymus very large, post-mortem.
44	9 weeks ...	Old pericellular fibrosis, with multilobular still showing signs of activity. Small vessels show marked irritation of endothelium.	Capsule thickened. Slight fibrosis. Vessels show fibrosis of outer coats, and activity of endothelium.	Not examined	+	....	Death 36 hours after onset, of strangulated hernia. Operation note: "Cæcum and appendix in sac—not injured beyond repair. Returned. Child greatly shocked." Post-mortem wound healthy, no peritonitis; no gangrene of contents of sac.
45	4 years ...	Pericellular fibrosis, but no vascular changes	Capsule thickened, fibrosis, and some changes, particularly in central arteries.	Not examined	— (ante-mort.)	....	Sarcoma of kidney.
46	1 year and 3 months.	Some increase in fibrous tissue. Vessels doubtful.	Capsule thickened. Fibrous framework decidedly increased. Vessels doubtful.	Not examined	Partial ...	....	Broncho-pneumonia. Death on 14th day.

No.	Age.	Histological Appearances.			Serum Reaction.	Mercurial Treatment.	Clinical History.
		Liver.	Spleen.	Bowel.			
47	10 months	Pericellular fibrosis. Some fibro-blastic activity in Glisson's capsules. Vascular changes, chiefly in larger vessels, which are thick-walled, and show active endothelium. No fatty change.	Capsule thickened. Slight increase in fibrous stroma, Some thickening of vessels with irritation of endothelium.	Ulceration of colon with mono-nuclear leucocytes in ulcerated area. Round cell accumulation in sub-mucosa. Vessels thick-walled. Endothelium active.	+	....	Bowel trouble since birth. 8 other children. Brother died, æt. 2 months. Waseermann +. Fatal attack of acute gastro-enteritis, lasting 1 week. No acute changes, post-mortem.
48	4 years and 6 months.	Multilobular fibrosis. Fatty degeneration. Vessels thick-walled, hyaline, with marked endarteritis.	Capsule thickened. Diffuse fibrosis. Vessel walls thickened and hyaline. Endothelium active.	Not examined	+	....	Diphtheria with retained tube. In hospital 6 months. Then contracted measles with bronchitis, and died on the second day.
49	4 months	Multilobular fibrosis of varying age. Vessels thick-walled, with endarteritis and periarteritis. Uniform engorgement. No fatty change.	Capsule greatly thickened. Diffuse fibrosis. Vessels as in liver.	Not examined	+	....	Cough for 5 weeks. Gastro-enteritis 4 days before death. 7 lb. weight. Death from malnutrition, no lesion being found in bowel, post-mortem.
50	4 months.	Fibro-fatty liver. Multilobular fibrosis. Vessels are doubtful.	Capsule greatly thickened. Marked fibrosis and thickening of coats of vessels which show endarteritis.	Ulceration of mucous membrane. Muscle thickened. Chronic inflammation.	Partial ...	....	Entero-colitis, 1 month's duration. No other children. No miscarriages.
51	7 months	Fibrosis—vessels thick-walled, endothelium active. Old multilobular and pericellular fibrosis.	Capsule thickened. Increase in fibrous framework. Vessels thickened. Endothelium active.	Chronic inflammation of colon and small bowel, with sub-acute exacerbation. Great ulceration. Chronic fibrosis of muscle coats. Vessels as in liver.	—	?	Entero-colitis of 3 months' duration. Clinically regarded as syphilitic. Nothing suggestive in family history.
52	10 months	Fibro-fatty. Multilobular and pericellular fibrosis. Foci of marked activity. Vessels show endarteritis and some fibrosis of outer coats.	Diffuse fibrosis. Vessels fibrosis of outer coats and activity of endothelium.	Chronic inflammation with engorgement of sub-mucous vessels, and fibrinous exudate on surface. Vessels thickened.	+	....	Entero-colitis.
53	2 months	Old fibrosis. Fatty liver. Vessels thickened with endarteritis and periarteritis.	Capsule thickened. Vessels thickened, and as in liver. Extreme diffuse fibrosis.	Old fibroid changes in outer coats. Sub-acute enteritis.	Partial ...	....	
54	6 weeks ...	Old multilobular fibrosis. Some increase of fibroblasts. Between columns of liver cells. Vessels thick-walled, with definite endarteritis.	Capsule thickened. Great increase in fibrous strome. Vessels thick-walled, with definite endarteritis.	Old fibrosis of muscle coat, with thick-walled vessels. Sub-acute inflammation.	+	....	Ill since birth. Undersized perionychia of right index finger. Enteritis. Broncho-pneumonia.

No.	Age.	Histological Appearances.			Serum Reaction.	Mercurial Treatment	Clinical History.
		Liver.	Spleen.	Bowel.			
55	6 months	Fibro-fatty, fibrous tissue, chiefly old. Vessels thick-walled. Endothelum active.	Not examined	Not examined	—	....	Broncho-pneumonia. Empyema, 2 months.
56	6 weeks.	Extreme fatty change. Great increase in fibrous tissue in Glisson's capsules. Patchy endothelial activity.	....	Some ulceration	—	....	Ill 3 weeks. Entero-colitis, but only slight lesions found post-mortem. Small, poorly-developed child.
57	5 months	Multilobular fibrosis. Pericellular fibroblasts. Vessels thick walled-with marked endarteritis. Some increase in fat.	Capsule thickened. Diffuse fibrosis. Vessels as in liver.	Sub-acute enteritis and colitis.	Partial	....	Entero-colitis. Ill for 2 months. No suspicion of syphilis clinically.
58	8 months	Fibrosis. Thick-walled vessels, with endarteritis.	Capsule thickened. Diffuse fibrosis. Vessels—fibrosis of outer coats.	Not examined	+ (ante-mortem.)	....	Ill 3 weeks. Colitis long continued. Mild inflammatory changes at post-mortem.
59	9 months	Fibro-fatty liver. Old fibrosis with more active areas. Smaller arteries are thick-walled with endarteritis.	Capsule slightly thickened. Slight fibrosis. Patchy endarteritis of vessels, which are thick-walled.	Not examined	+	....	Entero-colitis. Broncho-pneumonia. Slight mitral endocarditis, recent. Ill 3 weeks.
60	7 months	Capsule thickened. Increase of fibrous tissue in Glisson's capsules. Pericellular fibrosis active. Vessels thick-walled. Endothelium active	Capsule thickened. Diffuse fibrosis. Vessels as in liver.	Not examined	+	....	Erysipelas and general peritonitis. Ill 1 week. Chronic mild entero-colitis.
61	3 months	Multilobular and pericellular cirrhosis. Vessels thick-walled, with endarteritis.	Fine fibrosis. Vessels thick-walled. Endothelium active.	Not examined	—	—	Blue since birth. No anatomical lesion found, but heart dilated. Six other healthy children. No history of vomiting.
62	1 year and 7 months.	Capsule greatly thickened. Old multilobular fibrosis with activity. Some pericellular fibrosis. Vessels thick-walled. Endarteritis. No fat.	Capsule thickened. Marked diffuse fibrosis and vascular thickening with endarteritis.	Old chronic fibrosis in muscle coats. Ulceration of mucous membrane. Vessels as in liver. Terminal engorgement.	—	....	Entero-colitis. Acutely ill 7 days. No suspicion of syphilis clinically.
63	3 months	Extensive fatty change. Active fibroblastic infiltration in Glisson's capsules. Vessels show fibrosis of outer coats and, activity of endothelium.	Capsule thickened. Diffuse fibrosis, and vascular thickening with endarteritis.	Chronic fibrosis of muscle coats and mucous membrane; little glandular tissue remaining. Terminal engorgement. Vessels as in liver. Great overgrowth of endothelium in sub-peritoneal lymphatics.		....	Acutely ill 8 days. Entero-colitis. Colon showed evidence of chronic change at post-mortem.
64	1 year ...	Extreme fatty change. Old fibrosis with very active foci in Glisson's capsules. Vessels thick-walled. Periarteritis and endarteritis.	Capsules thickened. Diffuse fibrosis. Vessels as in liver.	....	Partial ...	....	Entero-colitis. Ill 9 days.

No.	Age.	Histological Appearances.			Serum Reaction.	Mercurial Treatment.	Clinical History.
		Liver.	Spleen.	Bowel.			
65	5 months	Extreme fatty change. Old fibrosis in Glisson's capsules of varying age. Vessels show great irritation of endothelium and some fibrosis of outer coats.	Capsule thickened. Fine fibrosis. Vessels as in liver. Some hyaline change in coats of central arteries.	Old chronic fibroid changes in sub-mucosa and muscle coats. Terminal engorgement.	+	Treatment with Hyd. c Cret. for 6 weeks.	Poorly nourished child. Regarded as syphilitic. Acutely ill 17 days. Death from entero-colitis. Family history not obtainable.
66	1 year and 6 months.	Fibrosis old and active. Vessels are not characteristic.	Capsule thickened. Fibrosis. Vessels show activity of endothelium and thickening of coats.	Not examined	—	....	Entero-colitis. Broncho-pneumonia.
67	3 years ...	Fibro fatty multilobular cirrhosis, with very active pericellular infiltration. Vessels thick-walled. Marked endarteritis.	Capsule thickened. Some increase in fibrous framework. Vessels as in liver.	Tubercular ulceration. Old fibroid condition. Vessels thick-walled. Endarteritis and periarteritis.	+	....	Measles and otorrhœa, followed by tubercular meningitis. Tubercular ulceration of bowel. Old tubercle at right apex. Interstitial keratitis.
68	5 months	Fibrosis, old and active. Vessels thickened. Marked endarteritis.	Capsule slightly thickened. Diffuse fibrosis. Vessels as in liver.	Not examined	+	....	
69	4 months	Fatty liver. Scarcely any normal cells now visible. Multilobular cirrhosis. Vessels thick-walled. Endothelium active.	Not examined	Colon—some ulceration. Marked catarrhal inflammation. Chronic catarrhal enteritis.	+	....	Gastro entero-colitis of 4 weeks' duration. Infant life child.
70	7 years and 10 months.	Fibrosis in Glisson's capsules and between cells, chiefly old. Vessels doubtful. Some increase in fat.	Thick capsule. Old fibrosis. Vessels thick-walled. Hyaline doubtful.	Not examined	—	....	Death from acute acidosis. No lesion found, post-mortem. Constipation and head-ache for 4 weeks. Urine contained diacetic acid and acetone.
71	5 months	Capsule thickened. Slight fibrosis. Marked fatty change. No active fibrosis. Vessels thick-walled, with marked endarteritis.	Capsule thickened Diffuse fibrosis. Definite endarteritis and fibrosis of outer coats of small arteries.	Some ulceration and sub-acute inflammation. Old fibrosis of muscle coat. Vessels as in spleen.	+	....	Ill 8 days. Convulsions, intestinal toxæmia.
72	8 months	Fibro-fatty. Great increase in fibrous tissue in Glisson's capsules. Marked atrophy of liver cells. Vessels are thick-walled. Endothelium active.	Not examined	Colon—ulceration of mucous membrane. Old fibrosis of muscle coat. Vessels as in liver.	+	....	Entero-colitis inter-mittent for 2 months. Traces of blood in motions at times.
73	7 months	Fibrosis pericellular and in Glisson's capsules—some very active foci. Vessels thick-walled. Endothelium active.	Diffuse fibrosis. Vessels as in liver.	Not examined	Partial ...	....	Broncho-pneumonia. Ill 1 day. Old history of diarrhœa and vomiting. 2 other children. 1 miscarriage.

No.	Age.	Histological Appearances.			Serum Reaction.	Mercurial Treatment.	Clinical History.
		Liver.	Spleen.	Bowel.			
74	I year ...	Intense pericellular fibrosis. Some increase in fibrous tissue of Glisson's capsules. Some fatty degeneration. Vessel walls thickened Fibrosis and endarteritis.	Diffuse fibrosis. Vessels as in liver.	Not examined	—	....	Scalds on face and forearm, not extensive. Died in 5 days. Chloroform for dressing, 3 times.
75	4 months	Fibro-fatty. Fibrosis, old in Glisson's capsules. No activity. Vessels thick-walled with active endothelium.	Capsule thickened. Great increase in fibrous framework. Vessels as in liver.	Old chronic entero-colitis, with sub-acute inflammation. Some ulceration.	Partial	....	Broncho-pneumonia En tero-colitis.
76	4 months	Capsule greatly thickened. Great increase in fibrous tissue of Glisson's capsule. Active pericellular fibrosis. Vessels thick-walled. Endothelium active.	Capsule thickened. Diffuse fibrosis. Vessels as in liver. Hæmorrhage and engorgement.	....	+	....	Broncho-pneumonia. Ill I day. Heart failure and sudden collapse. No family history obtainable.
77	10 months	Extreme fatty change, few normal cells persisting. Capsule thickened. Increase in fibrous tissue with active foci. Vessels show endarteritis and fibrosis of coats.	Capsule greatly thickened. Great increase in fibrous tissue of framework. Vessels very thick-walled with mark-endarteritis.	Not examined	+	....	Measles 2 months before death followed by entero-colitis and broncho-pneumonia. Was regarded as a congenital syphilitic. Family history not obtainable.
78	5 years ...	Fibrosis—chiefly multilobular with active foci of cell accumulasion. Vessels thick walled, hyaline Active endothelium	Some increase in fibrous framework. Vessels as in liver. Sub-capsular infarction.	Not examined	+	....	Ill I month. Lymphatic laukæmia. Death from broncho-pneumonia on second day.
79	7 months	Capsule not greatly thickened. Increase in fibrous tissue of Glisson's capsules with foci of round-celled activity. Vessels thick-walled. Endothelium very active.	Capsule thickened. Great fibrosis, chiefly old. Vessels thick-walled, with active endothelium.	Not examined	—	....	Ill 5 weeks, diarrhœa. Death from broncho-pneumonia. No lesion in bowel, post-mortem.
80	5 months	Fibro-fatty. Fibrosis of varying age. Small tubercles. Vessels everywhere show thickening of walls and endothelial activity.	Capsule thickened. Diffuse old fibrosis. Vessels as in liver. Miliary tubercles throughout the section.	Not examined.	+	....	Entero-colitis. Ill 8 weeks. Tubercular broncho-pneumonia. Generalised tubercle, post-mortem.
81	3 years ...	Slight interlobular fibrosis, chiefly old, with a few round cells. Vessels are not characteristic.	Some increase in fibrous framework old. Vessels are not thick walled.	Not examined	—	....	Ill I month. Olden endocarditis. Acute rheumatism and endocarditis. Heart failure.
82	4 years ...	Capsule thickened. Multilobular fibrosis of varying age. Active pericellular fibroblastic infiltration. Vessels thick-walled. Endothelium active.	Capsule thickened. Vessels show endarteritis and thickened hyaline walls.	Not examined	+	....	Ill 6 days. Started with boil on scalp. Marked albuminuria. Post-mortem, septic broncho-purulent, meningitis. Hæmorrhagic exudate over cordex of brain on left side, and some atheroma of aorta.

No.	Age.	Histological Appearances.			Serum Reaction.	Mercurial Treatment.	Clinical History.
		Liver.	Spleen.	Bowel.			
83	11 years ...	apsule thickened. Marked pericellular fibrosis, inactive. Vessels show endarteritis and fibrosis of outer coats.	Capsule thickened. Increase in fibrous stroma. Perivascular fibrosis. Vessels as in liver.	Not examined	+	....	Convulsions since infancy. Meningo - encephalitis. Ill 1 day. Considered to be syphilitic at post-mortem.
84	9 weeks ...	Active fibro-blastic change in Glisson's capsules and between cell columns. Fatty liver. Vessels—endarteritis and fibro-blastic infiltration of outer coats.	Not examined	Not examined	+	....	Gastritis and some enteritis for 1 week. Paralytic distension of the bowel. Post-mortem:— "Moderate colitis, considerable enteritis.
85	1 year and 7 months.	Capsule thickened. Fibrosis of varying age, chiefly multilobular. Some pericellular fibrosis. Vessels thick-walled. Some perivascular irritation and endarteritis.	Capsule slightly thickened. Increase in fibrous stroma, inactive. Vessels show endarteritis and hyaline walls.	Intense ulcerative colitis.	+	....	Diarrhœa and vomiting, intermittent for 6 weeks Some blood in stools.
86	1 year and 3 months.	Increase of fibrous tissue in Glisson's capsule. Inactive. Vessels show irritation of endothelium and thickened hyaline walls. No tubercle.	Capsule thickened. Some active cell infiltration of deeper layers. Vessels thick-walled, and hyaline. No tubercle	Not examined	Partial ...	....	Empyema. Ill 2 months first with tuberculous broncho-pneumonia.
87	13 years ...	Some increase in fibrous tissue around lobules. Vessels show some thickening of walls but no endothelial activity.	Slight fibrosis and thickening of capsule. No vascular change except in central arteries of malphigian bodies. In these, endothelium is active.	Not examined	—	....	Acute nephritis. Suppression of urine.
88	1 year ...	Active fibrosis, but no endothelial activity in smaller arteries. Great fatty change.	Not examined	Not examined	—	....	Ill 6 weeks. Diarrhœa, with slime and blood. Entero-colitis and broncho-pneumonia.
89	2 months	Capsule thickened. Great increase of fibrous tissue in Glisson's capsules. Vessels thick-walled and hyaline. Endothelium active. Great fatty change.	Great thickening of capsule. Vessels thick-walled and hyaline. Endothelium shows signs of irritation.	Not examined	Partial ...	Active treatment for 3 weeks.	Enteritis for 11 weeks. Malnutrition.
90	3 years and 9 months.	Capsule greatly thickened, old and recent fibrosis. Multilobular and pericellular vessels show fibrosis of coats, and endarteritis. Perivascular irritation.	Some increase in fibrous stroma. Vessels thick-walled, with active endothelium.	Old chronic fibroid change. Some ulceration of mucous membrane of colon.	=	....	Acute anæmia. Only child, no miscarriage.
91	1 year ...	Old and active fibrosis in Glisson's capsules. Some pericellular fibrosis. Vessels thick-walled. Endothelium active.	Slight increase in fibrous framework. Vessels as in liver, with marked periarterial irritation.	Not examined	—	....	

No.	Age.	Histological Appearances.			Serum Reaction.	Mercurial Treatment.	Clinical History.
		Liver.	Spleen.	Bowel.			
92	12 years ...	Multilobular and pericellular fibrosis. Vessels thick-walled, with hyaline change in walls. Endothelium active.	Capsule greatly thickened. Increase in fibrous stroma. Vessels as in liver.	Not examined	+	....	Ill 7 days, cough, vomiting, and epistaxis. Mitral regurgitation. No history of rheumatism. Post-mortem: Dilated heart, no valvular lesion. Slight pericarditis.
93	4 years ...	Old multilobular fibrosis. Very little evidence of activity. Old pericellular fibrosis. Vessels show endothelial activity, with thick hyaline walls.	Capsule thickened. Old fibrosis. Vessels thick-walled, hyaline. Endothelium of smaller vessels show signs of irritation.	Not examined	+	....	
94	2 years and 6 months.	Multilobular fibrosis of varying age. Vessels show activity of endothelium, fibrosis of outer coats, and perivascular irritation.	Capsule thickened. Diffuse fibrosis. Vessels as in liver.	Not examined	+	....	Ill 6 days, tubercular meningitis.
95	1 year ...	Fibrosis in Glisson's capsules and pericellular, inactive for most part. Vessels show endothelial activity.	Capsule slightly thickened. Increase of fibrous stroma. Vessels show activity of endothelium.	Not examined	Partial ...	....	Ill 6 weeks with measles. Temperature 100°. Broncho-pneumonia, but only slight lesions, post-mortem.
96	5 years ...	Capsule not greatly thickened. Multilobular cirrhosis of varying age. Active pericellular fibrosis. Vessels show endarteritis and fibrosis of outer coats.	Capsule thickened. Small vessels as in liver. Hyaline change in walls of larger vessels.	Not examined	+	....	Acute nephritis 10 days after discharge from Diphtheria Hospital; uræmic, 2 days.
97	3 years ...	Old and active fibrosis in Glisson's capsules and pericellular. Vessels thick-walled, with active endothelium. Some fatty accumulation.	Capsule thickened. Old diffuse fibrosis. Vessels as in liver.	Not examined	+	....	Pneumonia. Death on the third day. Temperature never above 100°.
98	7 years ...	Multilobular fibrosis, old and active. Vessels thick-walled, with endarteritis.	Great thickening of capsules and fibrosis. Vessels show marked changes as in liver.	Not examined	+	....	Was at school 4 days before death. Purulent meningitis. No focus found.
99	12 years ...	Extreme grade of multilobular cirrhosis, also some pericellular fibrosis. Vessels thick-walled, with endarteritis.	Capsule thickened. Great increase in fibrous stroma. Vessels thick-walled, and endothelium very active.	Not examined	+	....	Old standing cirrhosis of liver with ascites. No history of alcohol. Death from toxæmia 10 days after performance of an omentopexy.
100	7 years ...	Capsule greatly thickened, slight fibrosis and active fibro-blastic infiltration in Glisson's capsules. Vessels thick-walled. Endothelium active.	Capsule thickened. Some increase in fibrous framework. Vessels show activity of endothelium and fibro-blastic infiltration of outer coats. Some hæmorrhage.	....	—	....	Diphtheria. Death from toxæmia. No family history obtainable.

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